Journal

OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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Vol. 129 **OCTOBER 15, 1956** **No. 8**

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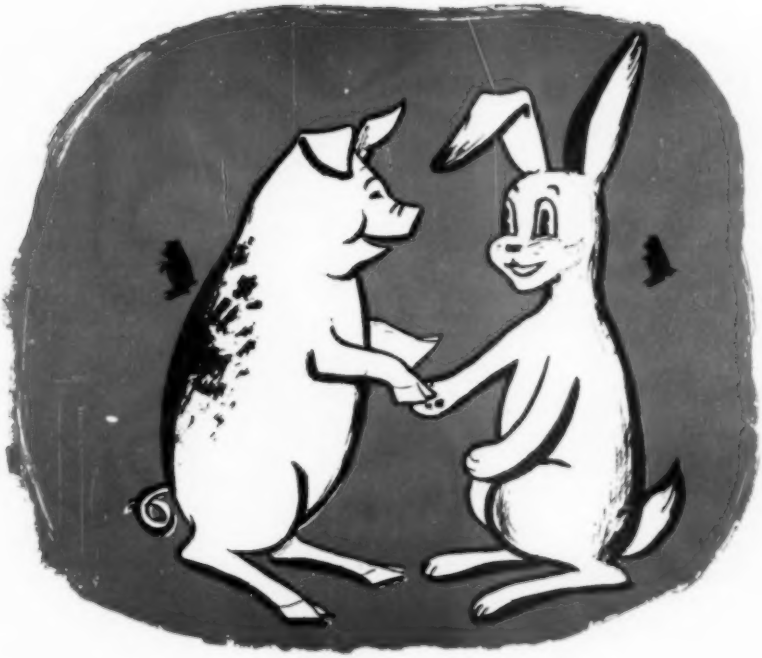

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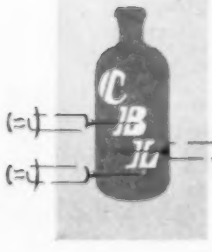

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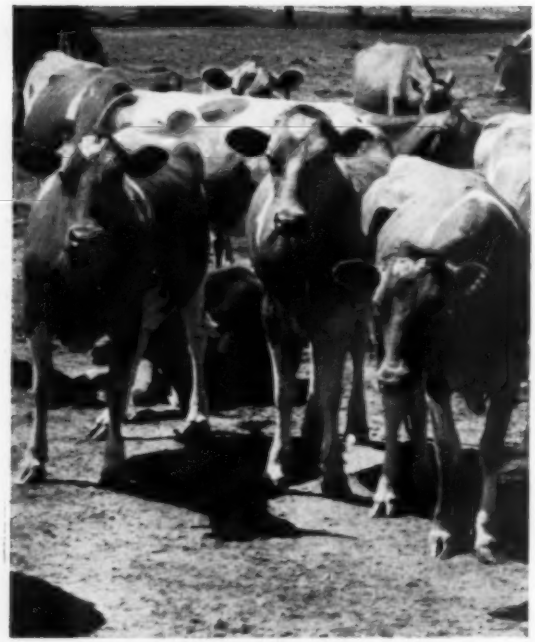

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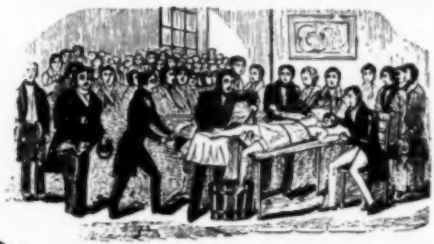

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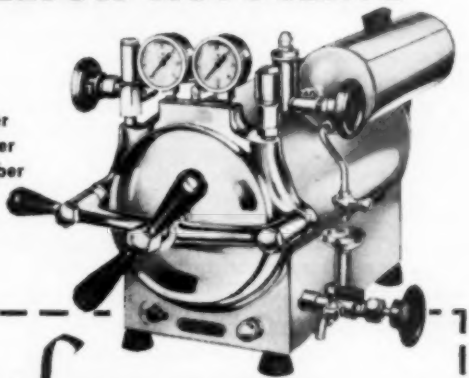

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News From Washington

The AVMA made known its views on legislation of special interest to the profession to several committees of the 84th Congress. **A representative of the AVMA testified on bills before seven committees (letters sufficed on five other occasions) in support of legislation** under consideration. In addition, numerous calls were made on AVMA legislative contacts for their support of bills backed by the Association.

★ ★ ★ ★

Bills likely to be introduced in the 85th Congress **of interest to the veterinary profession** will be those providing for U.S.D.A. appropriations for ARS (increased **funds** for **meat inspection);** compulsory **poultry inspection;** further **protection** against **diseases** of **livestock** and **poultry;** an advisory and research committee on **humane slaughter** of **livestock** and **poultry;** assignment of U.S.D.A. personnel to colleges and universities in a teaching capacity on request of an institution; an amendment to the Selective Service Act pertaining to medical, dental, and allied specialists who are regular registrants (doctor draft as such will be allowed to expire June 30, 1957); voluntary pension plans for self-employed individuals (Jenkins-Keogh Tax Deferment Plan).

★ ★ ★ ★

In an effort to overcome some of the obstacles concerned with the **tuberculosis-eradication program,** the chief of the Animal Disease Eradication Branch has asked the following persons to serve as an advisory group: Drs. Wm. A. Hagan, Wm. H. Feldman, Byron T. Olson, National Institute of Health; Jacob Traum, and Howard Johnson. The first meeting was held in Washington, D.C., on October 1.

★ ★ ★ ★

The Army Surgeon General has requested orders on 32 veterinarians to report for active duty in October; 20 are from the 1956 R.O.T.C. group who accepted commissions upon graduation, while 12 are direct appointments or had reserve commissions. This group will report to the Meat and Dairy Hygiene School on or about October 10.

★ ★ ★ ★

Three veterinarians were among a number of recipients of training grants in public health under the new program authorized by the 84th Congress (see JOURNAL, Sept. 15, 1956: adv. page 8).

★ ★ ★ ★

The **first two states to be certified** under the **accelerated brucellosis-eradication program** are Washington and Wisconsin. This milestone was celebrated recently in each state by a victory dinner. Among those participating at the respective celebrations were: the governors; Mr. Herman Aaberg, director, Livestock Department, American Farm Bureau Federation; and Dr. M. R. Clarkson, Deputy Administrator, ARS, U.S.D.A. The latter official was the principal speaker on each occasion. With Maine, New Hampshire, and North Carolina, there are now five states certified under the over-all brucellosis program.

★ ★ ★ ★

The **U.S.D.A.,** on September 6, **announced** a recent **amendment** to **federal regulations** governing **interstate shipment of animals,** which should aid in assuring the success of the brucellosis-eradication program.

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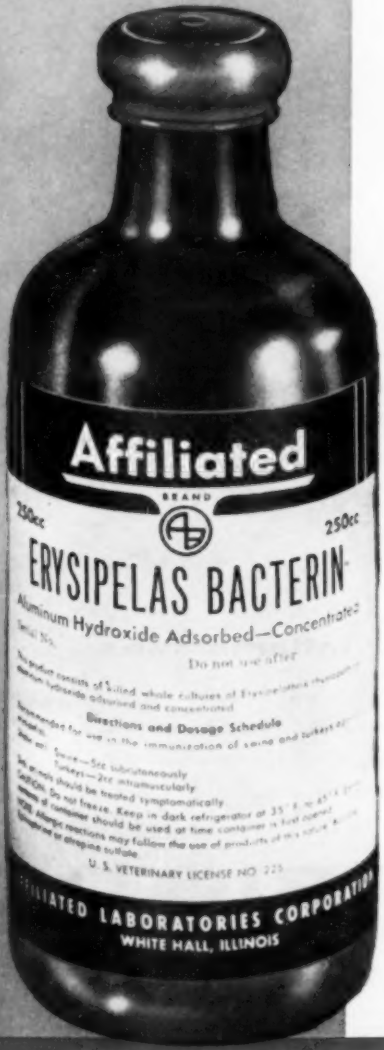

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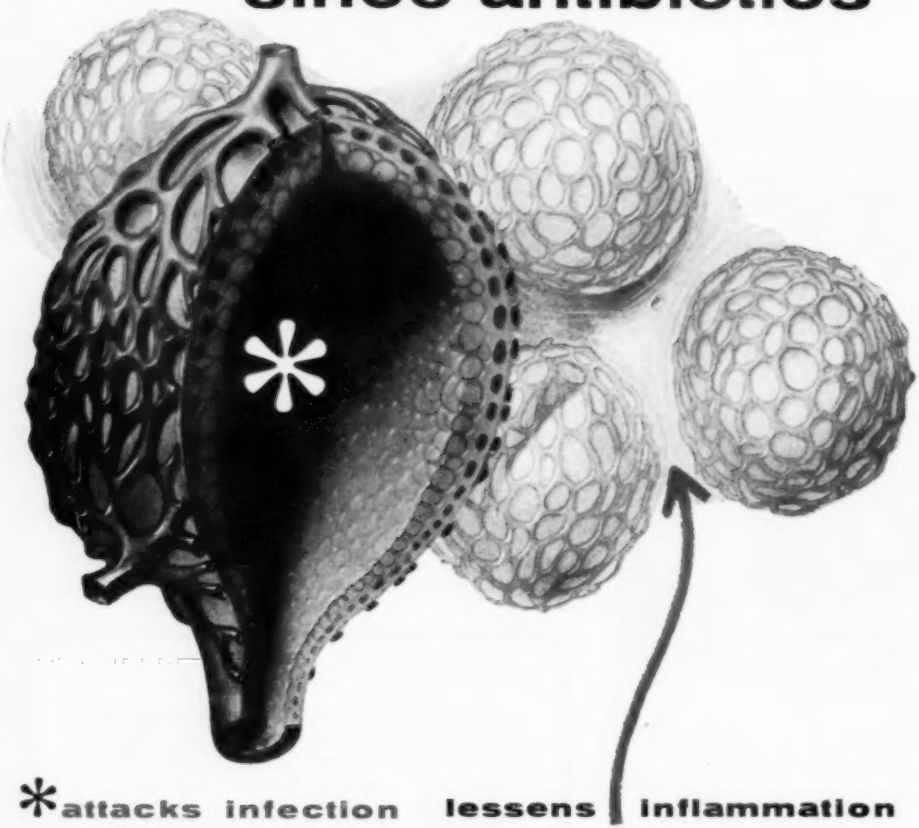

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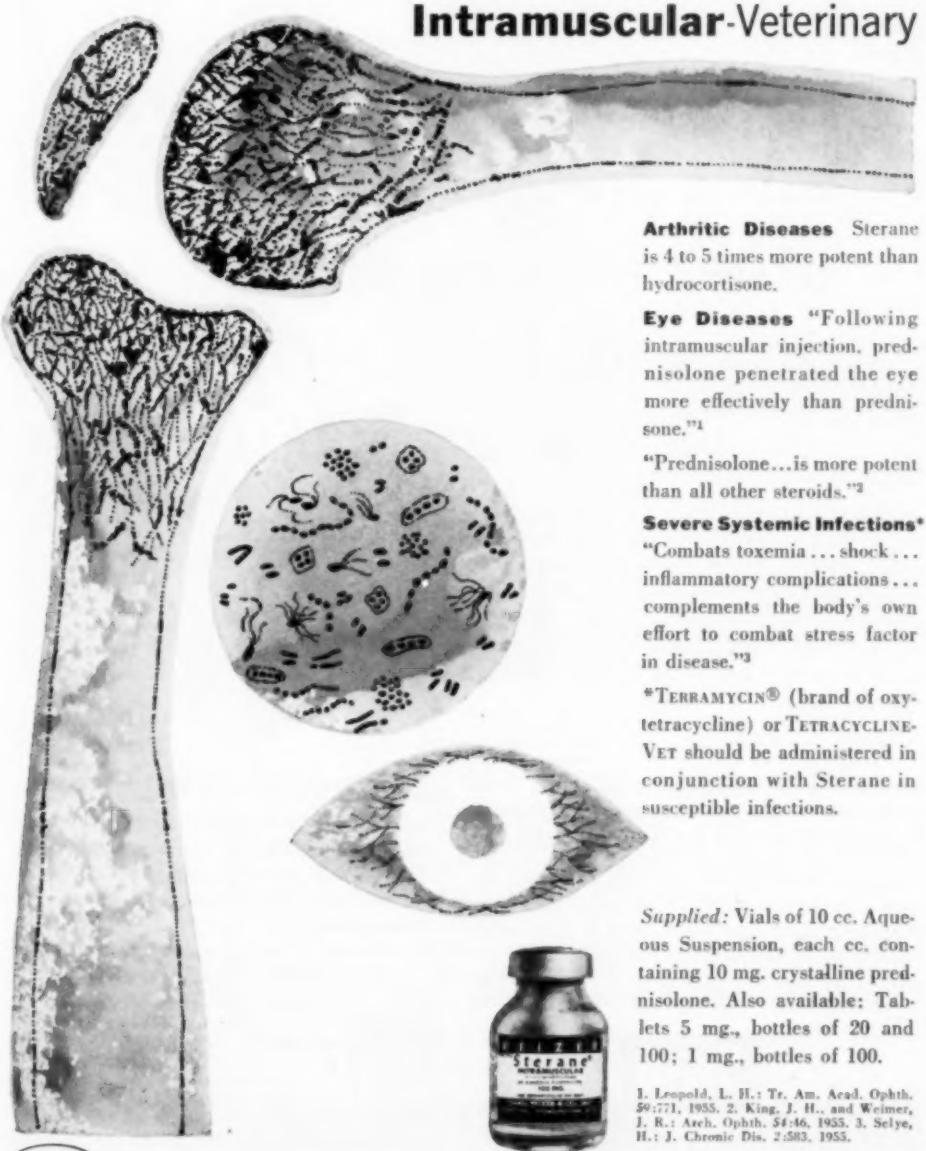

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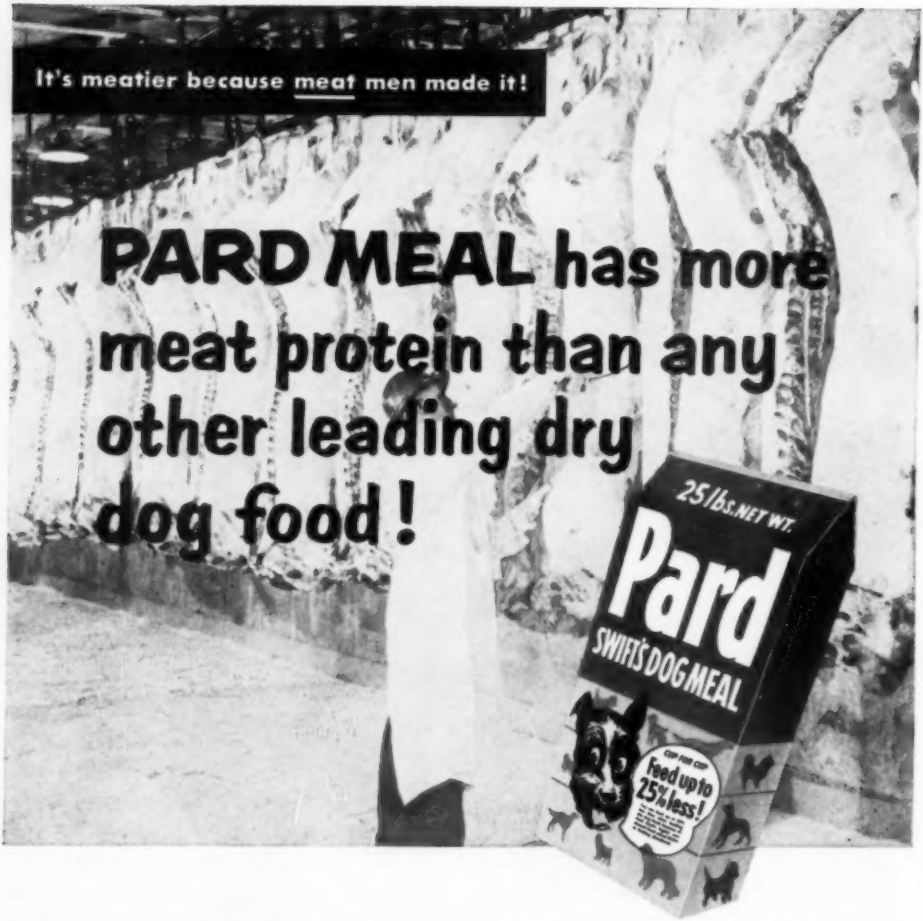
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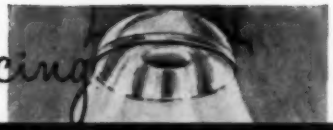

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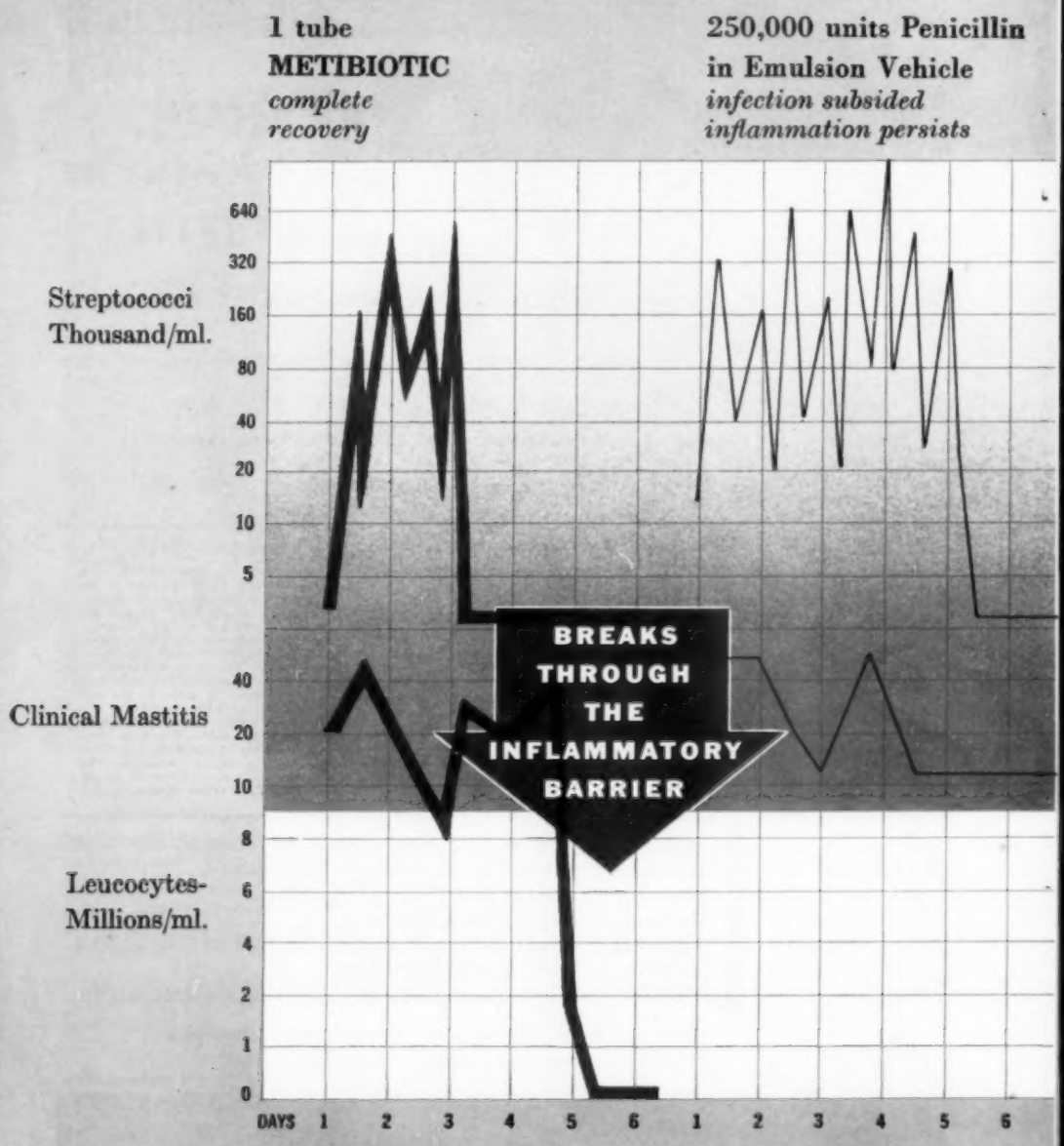

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1. Mosier, J. E.: Vet. M. 50:605, 1955. 2. Brookey, R. S.; Holt, S. H., and Siegel, D.: J. Michigan M. Soc. 54:805, 1955.

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JOURNAL
of the American Veterinary Medical Association
Chicago 5, Illinois
Vol. 129 OCTOBER 15, 1956 No. 8
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A Radiographic Study of the Effects of Mechanical Milking and Machine Vacuum on the Teat Structures of the Bovine Mammary Gland

A. C. PIER, D.V.M.; O. W. SCHALM, D.V.M., Ph.D.; T. J. HAGE, D.V.M.
Davis, California

THE MILKING MACHINE is potentially dangerous to mammary tissue. When its functional parts become worn and inefficient, or when it is negligently or improperly used, this device becomes a major contributor to mammary disease. It has been recommended[1] that when the percentage of cows with inflamed udders in a herd is high, then both the affected animals and the milking technique are in need of attention. However, many veterinarians feel they lack sufficient knowledge of the mechanics of this machine to advise on its use and, thus, an important segment of mastitis control programs is often neglected. It is the purpose of this article to discuss the action of the teat cup in milking and to point to situations in which the vacuum employed in mechanical milking may prove injurious to mammary tissue. A major part of this study was made possible by a newly developed technique which permitted radiography of the teat structures while the milking machine was in operation.

TEAT CUP CONSTRUCTION AND ACTION

The teat cup consists of a metal shell into which a cylindrical rubber liner is inserted (fig. 1). An airtight outer chamber, formed between the metal shell and the rubber liner, communicates with the pulsator mechanism through a side arm to which a rubber hose is attached. The pulsator alternately directs air into and draws it out from the outer chamber and this activates the rubber liner. When the teat cup is applied to the udder, the teat is sucked into the internal chamber of the rubber liner by negative pressure or "vacuum." The internal chamber of each of the four teat cups communicates with the milk "bucket" or pipeline through the "claw" arrangement. Since there is constant vacuum in the internal chamber of the rubber liner, the wall of the liner is forced to collapse against the teat when the pulsator directs atmospheric pressure into the outer chamber of the teat cup. The next instant, by pulsator action, a vacuum occurs in the outer chamber and now, with a vacuum on both sides of the rubber liner, the liner returns to its normal shape, the internal chamber is opened, and milk is drawn from the teat.

RADIOGRAPHIC TECHNIQUE

In order to visualize the action of the

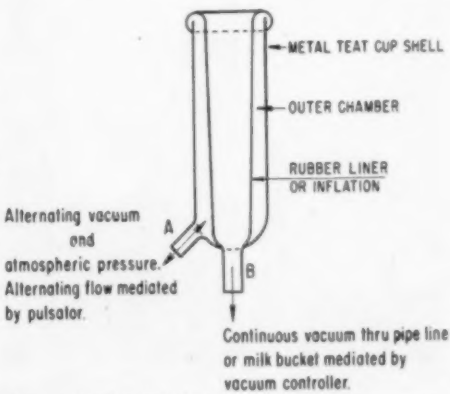
METAL TEAT CUP SHELL
OUTER CHAMBER
RUBBER LINER OR INFLATION
Alternating vacuum and atmospheric pressure. Alternating flow mediated by pulsator.
A
B
Continuous vacuum thru pipe line or milk bucket mediated by vacuum controller.

Fig. 1—Mechanical detail of teat cup construction.

From the School of Veterinary Medicine, University of California, Davis.

[1]Schalm, O. W., Pier, A. C., Gray, D. M., and Noorlander, D. O.: Application of the Whiteside Test to Bucket Milk and Foremilk in Herd Programs for the Prevention and Control of Mastitis. California Vet., (Jan.-Feb., 1956): 24-26.

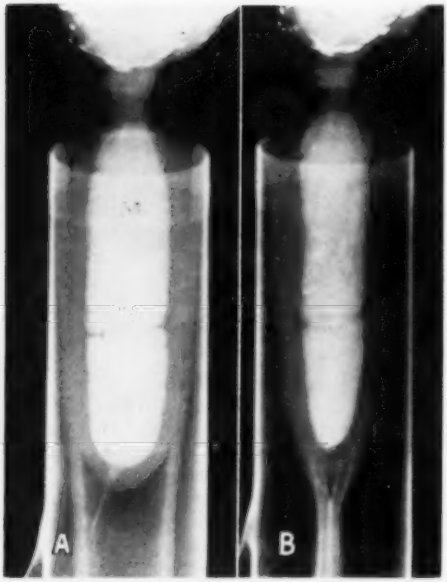

Fig. 2—Radiograph of teat cup and teat structures early in evacuation, (A) milking phase, (B) resting phase.

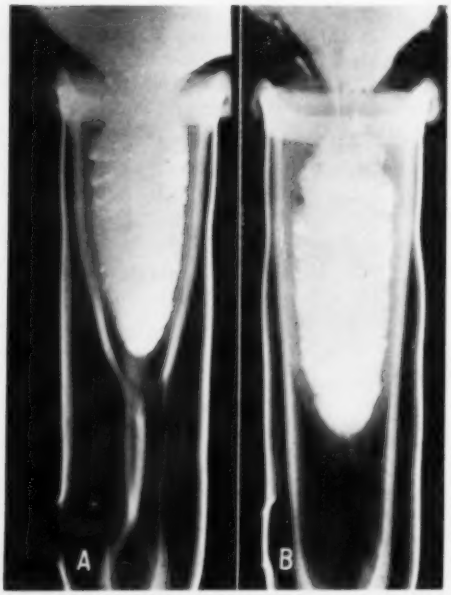

Fig. 3—Radiograph of teat and teat cup structures using large diameter liner, (A) resting phase, (B) milking phase.

rubber liner and vacuum on the teat structures, the x ray was employed to obtain radiographic pictures. The technique used consisted of infusing the unmilked udder with a suspension of colloidal barium and substituting a glass teat cup shell for the conventional metal one. The teat cups were then applied to the udder and a series of radiographs were made at various stages of milking. The exposures were made at 60 kv.p., 100 ma., at 1/40 of a second. Development of the technique for the making of motion pictures would provide a better insight into the sequence of events that occur throughout milking than can be obtained from periodically taken single exposures. However, the equipment for such work is not widely available.

DISCUSSION OF RADIOGRAPHS AND INCIDENTAL PHOTOGRAPHS

Radiographs (fig. 2) were taken in the early phase of milking and show both the milking and resting phases of teat cup action.

Figure 2A shows the milking phase. The

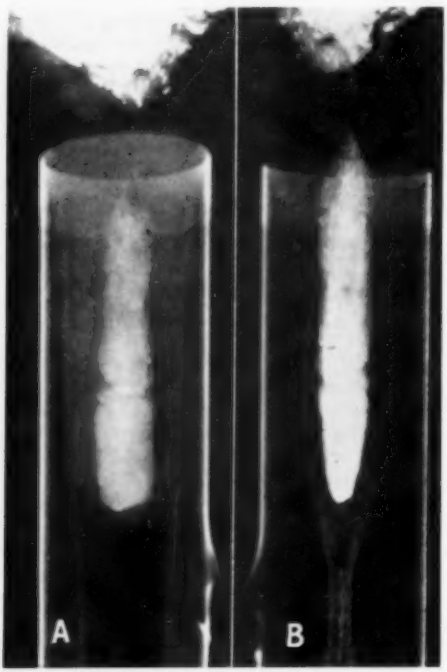

Fig. 4—Radiograph of teat and teat cup structures near end of evacuation, (A) milking phase, (B) resting phase.

rubber liner is expanded, *i.e.*, the internal chamber is opened, and milk is being drawn from the teat. At this time, there is a vacuum in both the inner and outer chambers of the teat cup. Thus, the stream of milk seen issuing from the teat is being sucked out by a vacuum rather than being expressed by external pressure as in hand milking. The teat sinus is seen freely communicating with the gland cistern immediately above it.

Figure 2B shows the resting or massaging phase. The rubber liner has collapsed below the end of the teat and is squeezing its tip. The degree of compression of the teat by the liner becomes progressively diminished from the orifice to the base of the teat. The function of the collapsing rubber liner is to massage the tissues of the teat to aid the circulation. Vacuum draws blood and tissue fluids in excess into the teat and, unless this were compensated for by an alternate action, the teat would become engorged and edematous. The complete collapse of the liner below the end of the teat does not lower the effective vacuum applied to the teat because rubber liners of large diameter tend to collapse in a figure-8 pattern, *i.e.*, they collapse completely at the center but leave channels at the margins of the collapsed portion. For this reason, liners of small diameter may be less injurious to the teat, since during the milking phase they tend to keep the vacuum confined to the end of the teat with a partial drop in vacuum during the resting phase.

Radiographs demonstrate the progressive constriction of the base of the teat from the beginning (fig. 3A) to the end (fig. 3B) of milking.

Early in milking, the positive pressure of the milk in the udder keeps the lumen of the base of the teat open so that milk flows readily from the gland cistern into the teat.

Figure 3A, taken during the resting phase, shows the partial constriction of the base of the teat caused by the teat cup, since the opening for receiving the teat is smaller than the diameter of the internal chamber of the liner. With the type of liner employed here, the teat balloons out to fill the internal chamber. Marked ballooning of the teat is prevented by liners of lesser diameter and, thus, less strain is produced on the tissues of the teat. Worn or deteriorated liners also permit an excessive ballooning due to increase in size of the liner (fig. 8) and loss of elasticity of the rubber. This excessive dilation of the teat may lead to additional stress on the sphincter tissues.

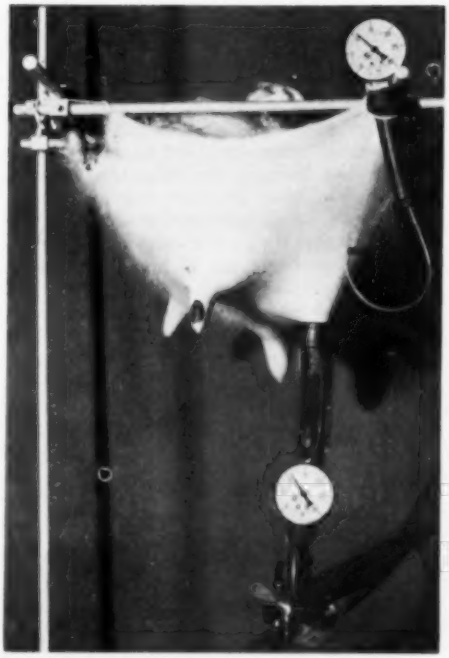

Fig. 5—Laboratory demonstration of the extension of vacuum to within the udder.

Figure 3B shows the milking phase late in evacuation. As milking proceeds, the cistern region of the udder tends to collapse,

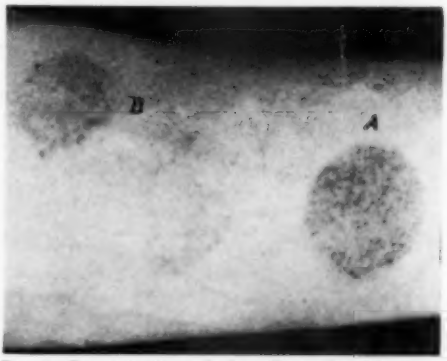

Fig. 6—Demonstration of the effect of milking machine vacuum on the skin of a human forearm, (A) immediate, (B) 48 hours later.

resulting in drawing more tissue into the teat cup and finally closing the lumen so that milk is no longer able to pass freely from the gland to the teat. When this, commonly referred to as "crawling" of the teat cup, occurs, the milker should pull down on the claw in order to re-establish the opening between the gland cistern and teat sinus so that the last milk or "strippings" can be quickly removed.

Radiographs (fig. 4) show the alterations in teat structure at the termination of mechanical milking.

Figure 4A shows the end of the milking phase when the tissue drawn into the teat cup is sufficient to completely obliterate the connection between gland and teat cavities. Some milk remains trapped in the gland cistern while the vacuum is extended into the teat cavity. The teat is considerably less dilated since the vacuum now affects it both internally and externally, and the teat wall is thickened due to stasis of blood and tissue fluids (compare fig. 4A with fig. 2A).

Figure 4B shows the resting phase when the action of the rubber liner is such that, with each pulsation, the tissues forming the internal wall of the base of the teat are caused to rub against each other in a kneading movement. As air rushes into the outer chamber of the teat cup, the liner collapses, compressing the teat upward; then, at the next instant, the liner expands and the tissues are again pulled deeper into the cup. When this massage effect is applied to the lymph and blood in the engorged teat tissues, the stress applied to the vessels is further increased. While the negative pressure in the teat cavity may fall with each collapse of the liner, it does not drop to zero. Therefore, when the teat cup is left in operation after milk flow

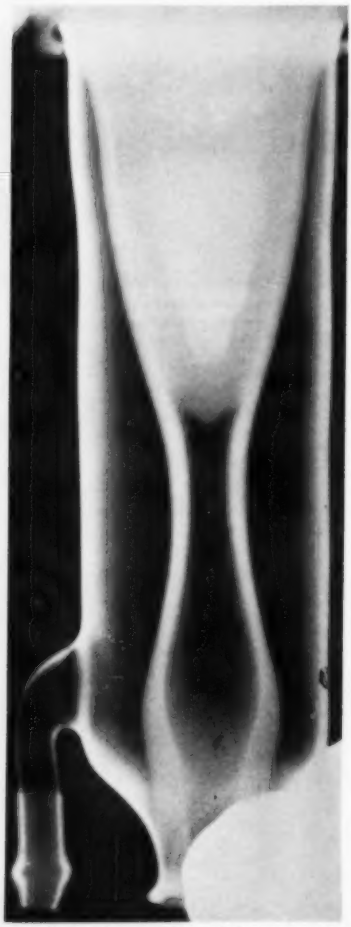

Fig. 7—Radiograph of teat and teat cup structures demonstrating the effect, during the milking phase, of perforated pulsator hoses or worn-out pulsator.

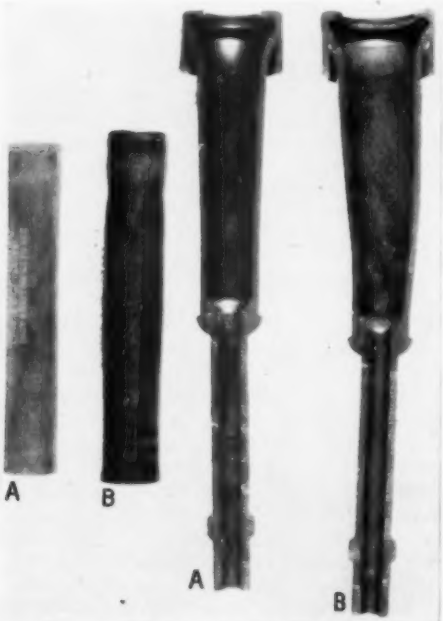

Fig. 8—Changes in size and shape of old liners (B) as compared with new liners of same type (A).

ceases, the internal tissues of the teat are subjected to continuous stress.

The laboratory demonstration (fig. 5) shows the extension of the vacuum to within the cavity of the teat. This udder was removed intact, when the cow was slaughtered, and was suspended from the frame as shown. A small plastic tube was inserted through an incision in the side of one mammary gland and was allowed to hang freely within the cistern. A teat cup with a glass shell, to permit observation of the liner, was placed on the teat. (This picture was taken as the liner collapsed during the resting phase.) As soon as the teat no longer contained milk, the vacuum affected the gland cavity through the teat orifice. The degree of vacuum within the udder, indicated by the upper gauge to which the plastic tube is connected, is essentially the same as that indicated by the lower gauge.

The effect on the human forearm of 12 inches of vacuum, unrelieved by massage, is shown (fig. 6). The teat cup was attached to the arm by the suction of the vacuum and was allowed to hang there for 45 seconds. A circular area of redness punctuated with petechial hemorrhages was produced (fig. 6A). Massaging the area with a finger caused the erythema to vanish but the petechial spots remained, some of them for two days (fig. 6B). For this demonstration, the rubber liner had been collapsing 48 times per minute but, since the moving portion of the liner was not in contact with the skin, massage did not occur. A similar effect might be anticipated on the internal tissues at the base of the teat when the machine is left in operation after milk flow has ceased.

A radiograph (fig. 7) depicts the effect on the action of the rubber liner when worn-out pulsators or leaky rubber hoses, connecting the pulsator with the outer chamber of the teat cup, are used. In these situations, the outer chamber of the teat cup has continuous, or partial, atmospheric pressure and, as a result, the liner remains partially or completely collapsed at all times.

A comparison is shown of the shape and size of worn-out rubber liners (fig. 8B) with the shape and size of the same liner when it is new (fig. 8A). With use, the rubber of the liner becomes impregnated with fat, leading to a loss in elasticity and an increase in the size of the liner. The worn-out liner is unable to respond effectively to the action of the pulsator and massage becomes ineffective. Furthermore, continuous action of the vacuum on the tissues of the teat is permitted and excessive ballooning of the teat with its accompanying stresses on the teat structure and mechanism ensue.

To maintain liners at peak efficiency, they should be removed from the metal shell daily, if feasible, and boiled in lye water (1 oz./gal.) for 15 minutes once every week. They should then be rinsed, dried, and stored in a dark, clean, cool place for a week while an alternate set of liners is used. Dairymen not wishing to trouble with boiling their liners, should discard them as soon as it becomes apparent that loss of elasticity is occurring. The length of time that a liner may be used, and remain effective, will depend largely on the number of cows milked daily.

SUMMARY AND CONCLUSIONS

This article portrays, by radiographic means, the action of the milking machine teat cup on the teat structures and draws attention to the fact that the negative pressure (vacuum) employed in mechanical milking is capable of injuring tissue when improperly applied. It shows what 12 inches of this vacuum can do to human skin in 45 seconds, indicating how the delicate internal tissues of the mammary gland can be injured when milking machines are allowed to remain on the udders after milking has been completed, or when machines with worn-out pulsators, perforated pulsator hoses, or deteriorated rubber liners are used. Experience has shown that milking equipment or milking methods, or both, are commonly at fault in dairy herds having a high incidence of mastitis. The veterinarian who is called upon to correct herd mastitis problems should have a thorough understanding of the milking machine and of what constitutes efficient operation and proper use.

Radiographic Study of Milking

Radiographic studies of the cow's milk-ejection mechanism, after contrast medium was injected into the teat, revealed that mechanical stimulation of the teat by squeezing was followed within a few seconds by a series of contractions which forced the contrast fluid to the roof of the

cistern and, eventually, into the larger milk ducts. These contractions were considered not to be associated with the milk ejection evoked by administration of oxytocin.—*Vet. Bull., July, 1956.*

A Preface to Veterinary History

A. BARTON, D.V.M.

Forest Hills, New York

The curious history of animal medicine would be disconcerting for an uninformed reader. Born in primitive civilizations, it was for a long time blended with human medicine until it was abruptly separated from it during the time of Socrates and Plato. For a few centuries it was held in high esteem at Rome and Byzantium, but then the light of its progress was dimmed and finally snuffed out and it was left to vegetate miserably, forgotten or disdained for 1,400 years.

In the recorded account of its vicissitudes will be noted the profound reasons of ostracism which struck not at individuals or groups but at the innocent art itself. Society, however, recognized the value or the necessity of that art.

With Plato, the West adopted a new philosophy. The soul was the thing that accounted for the dignity of man and aligned him with the gods. Since animals did not have souls, they were disassociated from humanity. They could not derive benefit from a divine art, such as medicine, unless they did so sacrilegiously.

Undoubtedly, Aesculapius welcomed and even deigned to answer those who pleaded the case for animals, but Hippocrates and his disciples were less tolerant. Grecian medicine was Platonic and man only was worthy of its assistance. Thus, for eight centuries, animal medicine was perpetuated alongside of human medicine and was practiced at Rome and Byzantium. Its precepts were purely Hippocratic. Its hippiatrists were trained men and often physicians, but they were forbidden entrance to the temple.

Possibly, with the aid of scepticism, the two medicines might have been brought together if an important event had not occurred. Christianity triumphed over the empire, and it felt the same remoteness for the animal as the disciples of Plato. An abyss separated the animal, a body without a soul, from man, who was created in the image of God and was the tabernacle of the immortal soul.

The policy of the Church emphasized the strictness of the doctrine. It was not only the question of implanting the abstract idea of the soul in the minds of the faithful, but also of fortifying itself against the dreaded influence of oriental religions, which Christianity did by expressing the obvious contradiction between man and beast. It was life and its mystery that was revered in Asiatic philosophy. Divine in principle, it sanctified all beings, and it might be the same everywhere today if Buddha had reigned over the world. To better affirm the absolutism of its dogma, the Christian church exaggerated its scorn of the animal by going so far as to refuse it the consolation of its pity.

Thus, the Platonic philosophy and the Christian religion were agreed in rejecting, as an outrage against the divinity, the idea of giving the animal the same care as man. Although the physician was no longer a preacher, he still remained a clergyman bound to a strict obedience. The control of the theologians was added to the discipline of Hippocrates since the Middle Ages. To be admitted to the universities, the only prerequisites demanded of the physician were that he speak the language of the clergy and affirm both his faith in the doctrine and his respect for the dogma before he was permitted to take the oath of admission.

Annihilated from the Christian world because of the interdictions hurled upon it, animal medicine was reborn as soon as it escaped the reprobations that weighed it down. With the Arabs, it found its place once more alongside of human medicine under the aegis of a religion that did not put any obstacles in its way. The art was taught in the great schools and the two medicines were equally respected. The influence that it exercised was so profound that it persisted long after the passing of the conquerors, and the Catholic kings accorded all their solicitude to the *albeiteria** of the Arabs.

It sometimes seemed as if the art of the Byzantine hippiatrists would be reborn on Christian ground. Moreover, since the

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Dr. Barton is a small animal practitioner in Forest Hills, N. Y.

Translated from the preface of Leclainche, E.: Histoire de la Médecine vétérinaire. Office du Livre, Toulouse, 1936.

*Albeiteria-veterinary surgery. Spanish. (Velasquez).

Italian Renaissance was influenced by the Arab world, the science of antiquity was revived and the book of Rufus spread it throughout Europe. It had only a fragmentary beginning there, for even though Salerne taught the postulates of Hippocrates, its teachers consented to dissect the pig in the absence of human cadavers, but the thought of studying animal disease seemed monstrous and sacrilegious to them.

At no time did the medical profession lend a helping hand to the veterinary art. When, from time to time, individuals with open minds showed some interest and tried to stimulate the art, they were rejected by their peers. Because he published an anatomy of the horse at the order of Charles IX, the physician Heroard was condemned and shunned by his colleagues of the faculty.

In the absence of any doctrine and any teaching, the practice of animal medicine was given over to the most flagrant form of quackery. Occasionally a teacher turned up who was cognizant of the study of the ancients, but he rarely attracted any disciples and his example was soon forgotten.

In the popular mind, the repugnance that the animal inspired was combined with the social aversion that attended animal physicians, and this was expressed in every country.

It was the philosophy of the eighteenth century that permitted the resurrection of veterinary medicine. When a philosophical justification of comparative anatomy and physiology was sought, it was found in the "philosophers of nature" who consecrated the redemption of the animal. Possibly, in spite of all the economic ramifications, veterinary education could not have been instituted elsewhere than in the atmosphere created by these new ideas. Possibly France owes as much to the encyclopedists as to Bourgelat for the creation for which he is honored.

One must not forget the insurmountable objections that Frederick of Prussia met in establishing the Berlin school. The Prussian medical college rendered its anticipated favorable opinion without enthusiasm, but it added, "We do not know how to ask the professors to mess around in the flesh of putrefying animals."

At the time of the inauguration of the school of Dresden, which permitted the dissection of the horse, the Duke of Courlande had to put his hand on the cadaver in order to overcome the repugnancy of his assistants. A few years later, the King of England had to enact severe penalties to protect the veterinary students who had to touch animal cadavers during the course of their study. None the less, popular prejudice persisted and Goethe later remarked that the establishment of the school at Jena brought out a contempt "that extended from the lowest to the middle classes."

It should be understood that until the end of the eighteenth century, the veterinary art, which had lain dormant since the fifth century of our era, existed without a doctrine and without doctors. It should also be understood that during this entire period its history was more social than medical. Freed from its original shortcomings, but not without a struggle, a century has sufficed to establish veterinary medicine in science and society. Now, after having attained these ends with so much difficulty, it is threatened with regression. It must adapt itself to changing conditions, for it is undoubtedly beginning a new period in its history.

[This final comment, made by Leclainche in 1936, referred to the low ebb of agriculture at that time and the difficulty in procuring students to fill the veterinary colleges. Schools used to send out circulars to prospective students enumerating the glowing opportunities in the veterinary profession. Since this time, the progress of the profession has been almost phenomenal and the challenge of the "threatened regression" has been satisfactorily met.]—Translator.

Establishing wild turkeys from New Mexico in the State of Washington will be attempted. Past attempts have failed chiefly because of the inability of the birds to survive during the winter. In exchange, New Mexico will receive blue grouse, chukars, or other game species.—*Sci. News Letter, Aug. 11, 1956.*

Medical Writing

Common errors in veterinary medical writing and suggestions for the preparation of articles and illustrations will be discussed in the JOURNAL at intervals (*see* editorial, this issue).

The AVMA published, over a year ago, a "Style Guide" for authors. A few copies are still available.

Surgery and Obstetrics
and Problems of Breeding

Fractures of the Neck of the Femur in the Dog
I. A Technique for Repair of Fractures of the Femoral Neck

A. J. CAWLEY, D.V.M.; JAMES ARCHIBALD, D.V.M., M.V.Sc.;
W. J. B. DITCHFIELD, D.V.M.

Guelph, Ontario

FRACTURES of the femoral neck in the dog and cat frequently result from automobile accidents. These fractures may involve either the intracapsular or the extracapsular portion of the neck.

Intracapsular fractures may occur through either the proximal femoral epiphysis (fig. 1) or the subcapital (proximal) portion of the femoral neck (fig. 2).

In epiphyseal fractures, the strength of the ligamentum teres appears to be most influential. It has been our experience that most femoral neck fractures are epiphyseal and that they occur in animals in which ossification of the epiphyseal cartilage is not complete. In most cases, the femoral head maintains its position within the acetabulum. On occasion, however, the ligamentum teres is stretched, and the femoral head is displaced in an anterodorsal position above the body of the ilium with its articular surface facing laterally. It seems reasonable to hypothesize that the tensile strength of the ligamentum teres is greater than that of the epiphyseal plate in these cases.

Subcapital and extracapsular fractures do not appear to be avulsion fractures like the epiphyseal type, but would seem to be more feasibly explained by the application of stress at right angles to the axis of the femoral neck, *i.e.*, such fractures would be likely to result if the femoral head remained in the acetabulum while the trochanter major moved forward, backward, or upward, relative to the position of the femoral head. The extracapsular or subcapital femoral neck fracture may occur in animals whether or not the epiphyseal plate is ossified. Presumably, these fractures result from an impact similar to that which causes luxation of the coxofemoral joint, the decisive factor being whether the angle of the blow will allow the femoral head to over-ride the acetabular rim.

Schroeder[1,2] outlined a method for closed reduction of extracapsular fractures by means of a modified Thomas splint in 1933, and a method for the correction of intracapsular fractures in 1939. This latter method involves the use of the modified Thomas splint plus two stainless steel pins placed at an acute angle through the trochanter major, the femoral neck, and into the femoral head. The two pins are secured externally by a bar. The use of a screw inserted through the trochanter major, the femoral neck, and into the femoral head was advocated in 1941.[3] The most frequent complications following this technique occur (1) when the drill bit or screw misses the head of the femur and enters the acetabulum; (2) when the drill bit breaks off.

In a modification of the technique for intracapsular fractures, described by Brinker,[4] two pins are driven into the femoral head through the trochanter major and neck and are fixed externally by a bar. The supporting Thomas splint is eliminated. This technique is used for both intracapsular and extracapsular fractures.

On the basis of 3 experimental dogs, a method of reduction by the use of Kirschner wires has been described, the goal being natural reduction of the fracture.[5]

A method using the fascia lata introduced through the neck and head of the femur and the acetabulum to immobilize the fractured neck while allowing continuity and mobility of the coxofemoral joint has been proposed.[6] It was considered relatively atraumatic, and is based on the replacement of the ligamentum teres, providing continued action of the hip while healing occurs.

These various techniques have their limitations. Since the fracture line is not visible to the surgeon, inadequate reduction or poor immobilization may result. In some instances, the blood supply to the femoral head is extensively damaged. These factors lead to fibrous, rather than bony, union of the fracture and delayed revascularization of the femoral head, the sequel to which is partial or complete aseptic necrosis of the head. The following technique was designed in an attempt to overcome these difficulties.

From the Ontario Veterinary College, Guelph.

TECHNIQUE

Exposure to the fracture line in fractures of the femoral neck is somewhat more difficult than exposure of fractures of other sites which are routinely pinned. The technique to be described is, however, simple enough to be used as a routine method in the repair of femoral neck fractures.

The technique of exposing the coxofemoral joint[7] consists of a skin incision extending from the mid-dorsal line to the stifle along the anterior border of the femur, and an incision of the underlying fascia lata along the same line. The vastus lateralis muscle is separated from the rectus femoris muscle, which lies anterior to it, by blunt dissection using the index finger. On the anterior surface of the vastus lateralis and vastus intermedius muscles, a branch of the anterior femoral artery and vein accompanied by a muscular branch of the femoral nerve are evident. These structures must be preserved. In intracapsular fractures, the joint capsule is opened at a point immediately posterior to the attachment of the rectus femoris muscle on the lateral surface of the ilium and the opening is extended around the circumference of the acetabular rim to achieve exposure of the femoral head (fig. 3). The freedom of movement of the femur depends on the extent of the opening in the joint capsule. It is not necessary to incise the joint capsule in the exposure of extracapsular fractures by this method.

Lateral rotation and traction of the femur exposes the fracture surface of the distal fragment. To view the entire fracture surface, it is necessary to retract the capsular attachment of the femur. In order to preserve the blood supply of the neck from the source, care is taken to avoid ex-

Fig. 1 (left)—The anteroventral view of one-half of the pelvis and the associated femur of a dog, showing an upper femoral epiphyseal separation. The femoral head is within the acetabulum and the neck displaced anterodorsal. The capsular attachment on the femur is shown distal to the epiphyseal plate.

Fig. 2 (right)—The anteroventral view of one-half of the pelvis and the associated femur of a dog, showing a fracture of the femoral neck. The line of fracture extends from the epiphyseal plate on the dorsum of the neck, ventrolateral to the capsular attachment.

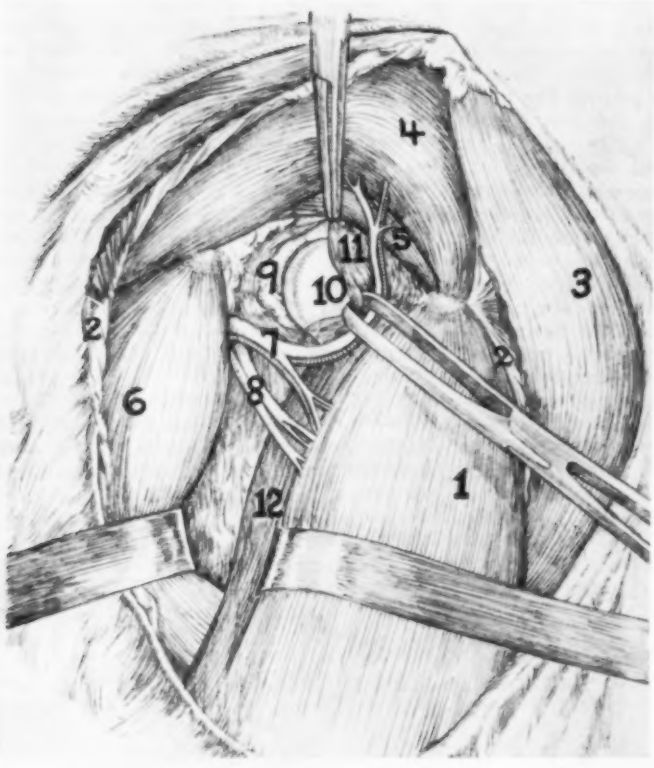

Fig. 3—A sketch of the exposed coxofemoral joint, showing the major structures in the area: (1) m. vastus lateralis, (2) m. tensor fascia lata, (3) m. biceps femoris, (4) m. gluteus medius, (5) m. gluteus profundus, (6) m. rectus femoris, (7) branches of anterior femoral artery and vein, (8) branch of the femoral nerve, (9) acetabulum, (10) femoral head, (11) joint capsule, (12) m. vastus intermedius.

Fig. 4—A radiograph of a femur of a dog, showing the pin introduced from the lateral aspect below the trochanter major, passed through the neck and embedded in the head at the same angle as the neck to the shaft.

cessive separation of the capsule from the neck of the femur. The fracture surfaces of both the distal and proximal fragments are carefully curetted. The unthreaded end of a 5/64- or 3/32-inch diameter, double-pointed Steinmann pin is introduced into the center of the fracture surface of the distal fragment and is passed through the neck across the shaft of the femur, using a hand-operated bone drill, and is then exteriorized at the distal end of the trochanter major. The pin is then withdrawn, again with the bone drill, from the lateral surface of the femur until its point is flush with the surface of the distal fragment. Reduction of the fracture is carried out and the threaded end of the pin is turned into the head until it is firmly embedded but does not penetrate the articular surface (fig. 4). The reduction must be as nearly perfect as possible, and the irregularities in the surfaces of both the proximal and distal fragments should fit into each other without twisting of the liga-

mentum teres. Movement of the femoral shaft should result in free movement of the femoral head without evidence of the pin scratching the acetabular surface. The pin is cut ½ inch beyond the lateral surface of the femur to facilitate later removal.

The joint capsule may be sutured, at the discretion of the surgeon, with fine (No. 000) chromic catgut. The sheaths of the vastus lateralis and rectus femoris muscles are not repaired. The fascia lata is sutured with fine catgut and the skin incision is closed. Postoperative radiographs are taken to determine the position of the fragments and the depth to which the pin was inserted. No additional support is applied to the affected leg and the animal is allowed to use the limb as it wishes, but extreme care must be exercised in handling the animal. Postoperatively, penicillin and streptomycin combined should be given intramuscularly daily for four or five days. Recovery is usually satisfactory. The pin is removed by way of a minute skin incision when the femoral neck is able to do without its support.

DISCUSSION

In undertaking repair of fractures of the femoral neck by the technique here described, consideration was given to (1) the simplicity of the operation, (2) the perfection of repair that can be achieved, (3) maintenance of the blood supply to the femoral neck and head, and (4) the fact that specialized equipment is not required.

Reduction and fixation of femoral neck fractures is a simple procedure provided both the head and neck of the femur are adequately exposed. However, experience is required to determine where and how extensively the joint capsule should be separated from the acetabular rim. With the femoral neck and head in view, the fracture is reduced. The only difficulty that may be encountered is the evasive movement of the femoral head in the acetabulum, but this is easily overcome by immobilizing the head with the tip of a closed pair of hemostats.

Partial interruption of the blood supply to both the femoral neck and head can be assumed to have occurred at the time of fracture, and some degree of aseptic necrosis will result. The surgical procedure should, therefore, interfere as little as possible with the remaining blood supply. Since the insertion of the pin through the femoral neck will result in some degree of vascular damage, the smallest possible pin should be used. On penetrating the femoral head, the pin goes somewhat dorsal to the fovea capitis and, therefore, does not interrupt the blood supply from the ligamentum teres.

Stability of the fracture line is insured by the pin inserted in the femoral head. Rotation of the head on the pin may appear possible, but it does not occur because of the two directional undulations of the epiphyseal plate or fracture surfaces. Because of this irregularity in the fracture surfaces of the head and neck, it is unnecessary to employ two pins in the fixation of such a fracture since a second pin merely contributes to trauma.

As observed in a series of 20 animals, the fracture line was firmly united by the fourth week after reduction and, at that time, the femoral neck showed some degree of aseptic necrosis. Radiographs taken at intervals showed osteoporosis of the neck followed by aseptic necrosis and progressive reossification that finally resulted in a femoral neck which the authors considered to be capable of normal weight bearing. In visually estimating the strength of the neck, consideration was given to the volume of new bone formed and the degree of density which was indicated by the radiographs. Some degree of flattening of the femoral head was noticed in 2 animals; reossification of the head and neck was relatively slow in both. It would appear that the speed of revascularization determines to some extent the changes observed in the articular surface of the femoral head.[8]

The fixation of such fractures results in joints which are much more functional than if repair is not carried out. The time elapsing between fracture and repair is important, since revascularization of the head and neck can not occur without reduction, impaction, and immobilization of the fragments. Rapid revascularization is necessary to the reossification process which prevents collapse of the devitalized proximal fragment. Once the bone becomes fragile, due to aseptic necrosis, the holding power of the pin is reduced and fixation is unsatisfactory.

The support afforded by the pin is necessary for revascularization and reossification in addition to weight bearing which can not be avoided in the dog. For this reason, and also because the difficulty of reduction is

increased after muscle shortening takes place, fractures of the femoral neck should be repaired as soon as possible, preferably within a week.

SUMMARY

A method of repair for fractures of the femoral neck with a single, double-pointed Steinmann pin is described. The results, based on 20 animals, indicate that efficient reduction with minimal trauma and early repair may be expected.

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Congenital Atresia of Colon of Calf

Congenital atresia of the distal colon is reported in 3 unrelated Aberdeen Angus calves, in England. Two had been normal for a week and were examined only after death, at 10 days of age. Each had acute peritonitis with multiple perforating ulcers at the termination of the markedly distended colon. In each, the rectum seemed normal except that it was represented by only a band of tissue where it joined the colon.

Surgery was performed on the third calf when it became dull and had a distended abdomen when 6 days old. Upon laparotomy, and after release of gas from the colon with a hypodermic needle, the atretic portion was resected and the colon anastomosed to the rectum about 9 inches from the anus and 3 inches from the pelvic reflection of the peritoneum. Recovery was rapid and, when 6 months old, the calf appeared normal.

Reference is made to an experiment where a mesenteric blood vessel was ligated in a pup, *in utero,* in which the results indicated that atresia, such as found in the calves, could be caused by interference with the mesenteric blood supply in the fetus.—*M. McClellen and J. A. Martin in Vet. Rec., July 14, 1956.*

Effect of X Ray on Egg Production

X-irradiation of the exposed oviduct of hens resulted in the production of malformed eggs similar to those laid following respiratory disease or the partial resection of the oviduct. Since such irradiation produced no pathological changes in the other organs of the body, this suggests that the peculiar eggs produced following a respiratory disease are due to the effect on the oviduct and are not secondary to changes in other organs.

The oviduct seemed to be relatively radioresistant since it remained functional after dosages up to 5,000 r, whereas a similar dosage to the whole body would kill many birds in 30 days.—*Poult. Sci., May, 1956.*

X-Ray Irradiation and Fetal Damage.—A woman with carcinoma was given roentgen irradiation (600 r to each of 2 abdominal fields and 300 r to the lower back) without knowing that she was four months pregnant. The 4-lb. child was born normally but later was found to have microcephaly, cerebral atrophy, idiocy, retarded growth, and other anomalies. Irradiation of the pregnant uterus is known to produce defects in the offspring, especially in the central nervous system. The nerve cells in the embryo are particularly susceptible to ray damage.—*J.Am.M.A., July 21, 1956.*

Purulent Pericarditis

Before the advent of antibiotics, the mortality for untreated cases of purulent pericarditis in man was about 100 per cent, 50 per cent when pericardiostomy was performed. It is now successfully treated with repeated paracentesis plus antibiotics, locally or parenterally, with surgical drainage when these measures are ineffective. Irrigation of the pericardial sac with streptokinase and streptodornase, to liquefy the pus, facilitates drainage.—*Am. J. Surg., June, 1956.*

Inguinal Metrocele (Gravid) in a Bitch

RODERICK SNOW, D.V.M.

Reseda, California

While inguinal hernias are not uncommon in middle-aged and aged unspayed bitches,[1,2] inguinal metrocele with a full-term fetus *in utero* is apparently an unusual phenomenon.

In the following case report, the herniation of the uterus must have occurred before or during an earlier stage of fetal development since the diameter of the expanded, but intact, inguinal rings could not have permitted passage of the fully developed fetus.

CASE REPORT

A 6-year-old female Cocker Spaniel was brought to the hospital on June 27, 1956, with a pendulous mass about 12 cm. in diameter protruding from the right inguinal region. The mass was apparently painless, was not reducible, and was first thought to be a large posterior mammary tumor. The animal had a normal temperature, good appetite and elimination, and was in good general condition. There was a thick, greenish vaginal discharge typical of that seen at parturition.

On closer examination, the mass appeared to have a dense central area surrounded by fluid and soft tissue, suggestive of a fetus, and this was confirmed by x-ray examination (fig. 1).

The owner was unaware of the pregnancy and had learned of the hernia only two weeks previously when the dog was groomed.

Under pentobarbital anesthesia, and after the usual preoperative preparations, the skin was incised, exposing a large hernial sac. The contents proved to be most of the internal genitalia including the ovaries, the horns and part of the body of the uterus, and the elongated ligaments. One horn contained the fetus which was dead.

The body of the uterus, together with its blood vessels, was ligated with chromic catgut as close to the external ring as possible.

Dr. Snow is a practitioner at the Reseda Veterinary Hospital, Reseda, Calif.

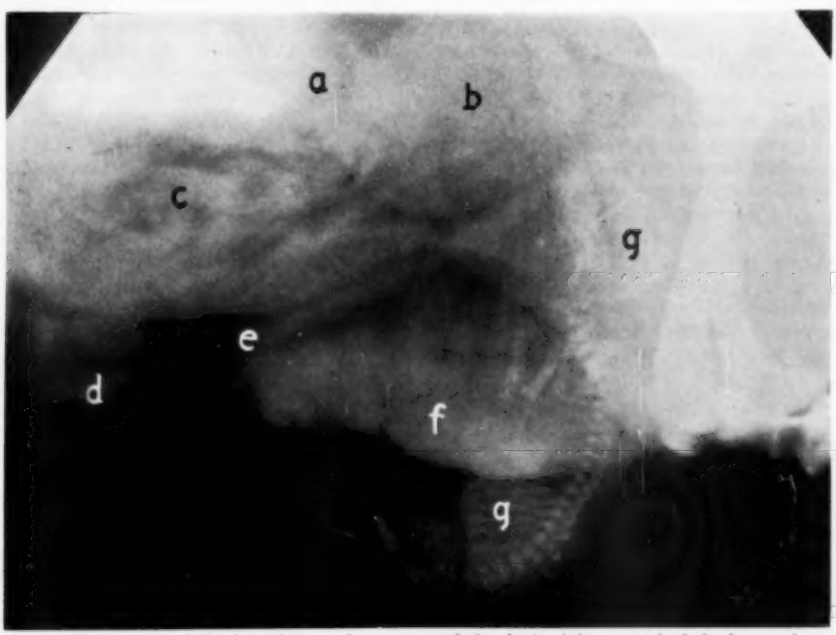

Fig. 1—Radiograph of the lateral view of a portion of the dog's abdomen and of the hernia showing: (a) kidney; (b) urinary bladder; (c) small intestines; (d) nipple; (e) abdominal wall; (f) mammary gland; and (g) fetus in the hernial sac.

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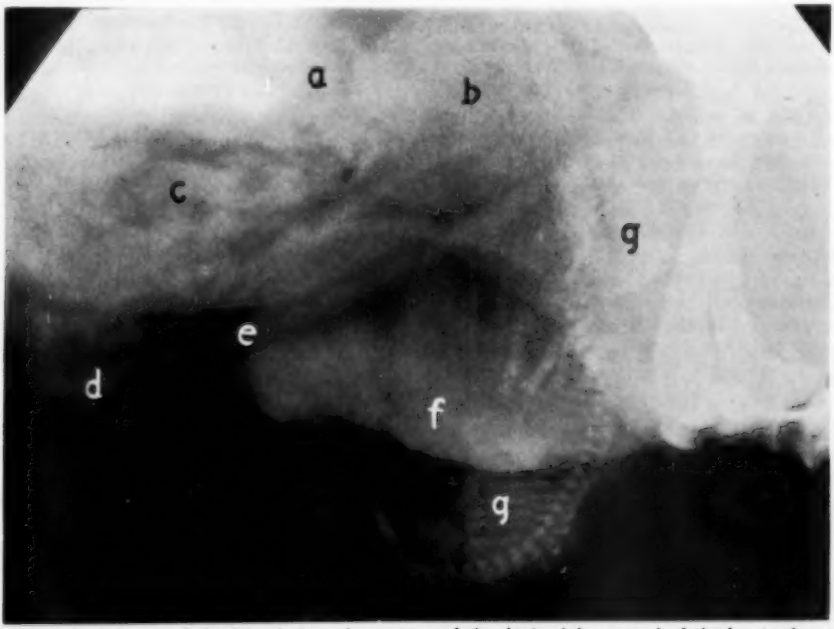

Fig. 1—Radiograph of the lateral view of a portion of the dog's abdomen and of the hernia showing: (a) kidney; (b) urinary bladder; (c) small intestines; (d) nipple; (e) abdominal wall; (f) mammary gland; and (g) fetus in the hernial sac.

The utero-ovarian vessels were then ligated and the intact genital organs were excised.

The hernial sac was excised at the edges of the dilated external ring and the peritoneal borders of the inguinal opening were approximated with a double row of continuous chromic catgut sutures. The subcutaneous pocket was obliterated as nearly as possible with continuous catgut sutures and, after excising the redundant portions of stretched skin, the cutaneous closure was made with interrupted sutures. A sterile pressure pack was bandaged over the wound.

When opened, the gravid uterus revealed a normally developed fetus in the early stages of decomposition.

SUMMARY

An inguinal, gravid metrocele in a mature bitch was relieved by an extrainguinal panhysterectomy and surgical repair of the inguinal hernia. Duration of the hernia was unknown, but much of the development of the full-term, dead fetus had occurred after herniation.

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[Inguinal hernias in dogs are usually metroceles in middle-aged, or older, whole females. The entire uterus and often the ovaries may be present in the sac. A different type of gravid metrocele was seen, in 1938, in a Fox Terrier which had been run over by a car several months previously. Following the accident, she had periodic episodes of dyspnea and cyanosis. At term, she delivered 3 puppies after which her respiratory disability increased. She died the next day in one of these paroxysms. At necropsy, a left side diaphragmatic hernia was found with the left uterine horn and ovary extending through the ring into the thorax. The herniated portion of the uterus contained a full-term fetus too large to be expelled through the diaphragmatic ring. —W.H.R.]

Relaxin—the Third Ovarian Hormone.—Relaxin, a nonsteroid hormone identified as producing relaxation of the symphysis pubis in the estrogen-primed guinea pig, was given intramuscularly or intravenously to 78 patients and volunteers with no evidence of sensivity. Results were gratifying in 15 patients in active labor, with response in 60 to 90 minutes regardless of the route of administration. Patients with certain types of disorganized uterine contraction (inertia) responded with normal labor and rapid delivery. The number of patients is too small to draw conclusions.—*J.Am.M.A., July 21, 1956.*

Potential Services of a Bull

Data on a 2,550-lb. Holstein-Friesian bull, 5 years old, from which semen ejaculations were taken three times weekly for four years, show that the volume averaged 2.8 ml. the first year, 3.8 the second, 5.0 the third, and 6.0 ml. the fourth year. When the spermatozoa content was computed, this indicated potential services of 52,289 the first year up to 119,676 the fourth year. The bull was maintained on a 14 per cent protein ration with exercise restricted to his 8- by 12-ft. box stall and going to the collection stall 20 ft. away three times per week.—*J. Dai. Sci., July, 1956.*

Control of Estrus with Progesterone

In research with beef heifers at the University of California, injection of progesterone prevented ovulation for 15 to 23 days, regardless of the stage of the cycle. When a single implant of crystalline progesterone (540 to 1,120 mg.) was given, ovulation occurred during the following estrus in only 95 per cent of the heifers. When this implant was followed, in 15 days, by an injection of 2,140 I. U. of equine gonadotrophin (in an attempt to increase ovulation), 16 of the 19 heifers came in estrus one to four days after the latter injection. All of them ovulated; however, only 17 per cent conceived after insemination.—*J. Anim. Sci., Aug., 1956.*

Fetal Membrane of an Elephant.—Four months after a female elephant was captured in Ceylon, she aborted a fetus (5 in., crown-rump) which was presumed to be at the four-month stage, since the dam was showing signs of estrus when captured. The fetal membrane (wt. 1.5 lb.) had three cotyledons, two of which were elongated (about 2 by 8 in.) and the third was kidney-shaped (1.7 by 3.6 in.). The gestation period of elephants is about 18 months. *Ceylon Vet. J., March, 1956.*

Hemolytic Anemia in Baby Pigs – Report of a Case

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HEMOLYTIC ANEMIA in newborn foals has been thoroughly investigated.[1,2] The condition was found to occur when mares, sensitized to certain types of erythrocytes, were bred to stallions which transmitted that type of blood cell to their offspring. Icterus developed in an apparently normal foal after it had obtained the specific erythrocyte-destroying antibodies from the dam's milk. A similar, naturally occurring condition has been described in newborn mules.[3,4] A similar disease has been experimentally induced in dogs.[5]

An isohemolytic disease in pigs was experimentally produced by injecting pregnant gilts with erythrocytes to the boar to which they were bred.[6] Recent excellent work[7] has resulted in the identification of two basic blood groups in swine (A and O), and it has been established[8] that crystal violet hog cholera vaccine can stimulate production of erythrocyte iso-antibodies in pigs. In a recent survey,[9] it was also found that detectable levels of serum iso-antibodies were confined, almost exclusively, to the vaccinated herds.

An incidence of hemolytic disease in baby pigs has been reported in the United States.[10] In this case, the sows had received crystal violet hog cholera vaccine in the routine immunization program. A report[9] on the naturally occurring disease in pigs in Great Britain suggests that it is, perhaps, incorrect to classify the disease as a naturally occurring phenomenon until iso-immunization can be shown in the absence of vaccination.

The purpose of this paper is to report a second incidence of naturally occurring hemolytic anemia of baby pigs in the United States. The diagnosis of iso-hemolytic disease is based principally on clinical observations, pathological findings, and on limited hematological observations.

CLINICAL HISTORY

From August, 1955, to March, 1956, 38 sows, all of which had been bred to a single boar, farrowed litters of pigs which appeared normal at birth. In more than half of the litters, a majority of the pigs developed varying degrees of icterus and died when 1 to 3 days old. In two litters, all of the pigs died but a few pigs in each litter were unaffected. In several instances, affected pigs which were transferred to foster mothers rapidly recovered. A total of approximately 75 newborn pigs died during this nine-month period.

The sows were mixed breeds of Hampshire, Duroc Jersey, and Landrace. The boar was a crossbred Hampshire-Duroc Jersey. Six of the sows were known to have previously farrowed one or more litters that were similarly affected. All animals on this farm were rountinely vaccinated, at six-month intervals, with a tissue hog cholera vaccine of swine spleen origin. It must be assumed that considerable quantities of blood are present in this type of vaccine.

PATHOLOGICAL FINDINGS

Several pigs which had recently died were examined and all showed similar pathological changes. Generalized icterus of varying severity was present. Focal and diffuse hemorrhages were scattered throughout the viscera, and a considerable quantity of sanguineous fluid was present in both the pleural and peritoneal cavities. The spleen and liver were slightly enlarged, apparently from congestion. Extremely fine mottling of a lobular pattern was present in the liver. In all instances, the stomach was filled with recently ingested milk.

Microscopic examination of blood smears taken before death from an affected pig revealed marked dyscrasia, characterized by the presence of immature erythrocytes and bizarre alterations in the shape (poikilocytosis) and size (anisocytosis) of the more mature forms.

Cultures of several organs of each pig developed no pathogenic bacteria. Serological blood tests, for *Leptospira pomona*, given to the sire and several affected sows, were negative.

Histological examination of the liver revealed marked congestion and slight-to-moderate erythropoiesis within the sinu-

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soids. The architecture of the hepatic cords remained unaltered. The capillary bed of the spleen was congested, and early diffusion of the lymphoid follicles was evident. There were moderate reticuloendothelial proliferation and numerous foci of erythropoiesis in the red pulp.

Only a limited number of serological tests were possible. Cross-matching tests were performed wherever possible. Equal volumes of serum, diluted 1:2 with physiological saline solution, and a 50 per cent suspension of washed erythrocytes were used in each instance.

Agglutination occurred when serum from a sow which farrowed an affected litter, in a dilution of 1:40 or 1:60, was mixed with washed erythrocytes from each of 2 pigs which had recovered. The same results were obtained when serum in a dilution of 1:80, from the same sow, was mixed with erythrocytes of the boar. Agglutination failed to occur in any dilution when boar serum was mixed with washed erythrocytes of 2 pigs which had recovered when placed with foster mothers, or with erythrocytes of the 3 sows which had farrowed affected litters. This suggests that the blood of the boar and 2 recovered pigs were compatible, whereas there was some degree of incompatibility between the blood of the boar and that of the 3 sows tested.

DISCUSSION

There is a gap in our knowledge concerning swine blood groups and possible incompatibilities, probably because of limited interest, since few clinically recognized cases have been reported. Although it was not possible to do detailed serological studies of this case, it is felt that the clinical, histological, and the limited hematological evidence justifies a probable diagnosis of isohemolytic disease.

There is strong evidence that erythrocytes of the affected pigs were destroyed by an agent present in the colostrum of the sow. This point is supported by the recovery of a majority of the affected pigs that were removed promptly after birth from the source of the erythrocyte-destroying agent present in the colostrum of the dam. The mechanism by which the sows, in this case, were immunized against the erythrocytes of their baby pigs can only be theorized. If the pattern follows that of the horse, then it might be postulated that there was iso-immunization of the sow to certain blood types present in the unborn pigs. In this event, the blood type of the sire would differ from that of the dam and only those pigs with a type comparable to that of the sire would be affected. This would, then, suggest the possibility that iso-immunization occurred *in utero* in much the same manner as it does in man. Thus far, this phenomenon has not been proved in swine.

It has been suggested[6] that the injection of hog cholera virus made from whole blood might sensitize a pregnant sow and cause her to produce iso-immune pigs. The iso-antibody titer produced in hogs by injection with crystal violet vaccine depends primarily upon the number of vaccinations which a given animal receives.[8] Vaccines used in the present case report were prepared from suspensions of spleen tissue. It is reasonable to assume that the blood contained in spleen suspensions might stimulate the production of iso-antibodies as does crystal violet whole blood vaccine. This raises a question of considerable interest and perhaps of practical importance regarding the possibility of immunizing sows against certain blood types by repeated injections of any vaccine containing whole hog blood or, more particularly, swine erythrocytes.

A majority of the pigs in the case reported here exhibited some degree of icterus, a clinical sign not observed in a case reported previously.[10] The development of icterus depends upon the duration and severity of the clinical disease. Icterus is not likely to occur in an acute, fatal hemolytic crisis. By contrast, a prolonged clinical course allows time for tissues to become saturated with bile pigment. When sudden, unexpected deaths occur in newborn pigs, with or without clinical icterus, iso-hemolytic disease might well be considered.

SUMMARY

A case of naturally occurring hemolytic anemia in baby pigs is reported. A diagnosis of iso-hemolytic disease was projected principally on clinical, histological, and hematological grounds. The condition continued to occur over a period of nine months, involved a total of 23 litters from 17 sows, and resulted in the loss of approximately 75 pigs. A majority of the affected pigs recovered when transferred, immediately after birth, to foster mothers,

indicating that the erythrocyte-destroying agent was present in the colostrum of the dam. Several mechanisms which might play a part in the production of hemolytic anemia in baby pigs are discussed.

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Hemolytic Anemia in a Litter of Newborn Pigs

On April 15, 1932, a Hampshire sow farrowed 9 healthy appearing, active pigs, her second litter. About three hours later, all were still normal and nursing but they soon became weak and none lived more than six hours. Necropsy of 5 pigs revealed no normal blood, just a reddish watery fluid with the erythrocytes precipitated into blackish clots. The livers and kidneys were blue-black and the spleens were almost black. The sow remained normal. She had been vaccinated with cholera serum and virus five months previously.—W.A.A.

The American Landrace Association held its first national breed conference at Springfield, Ill., in August.

Remarks on Shipping Fever in Canada

Confusion arises when shipping fever is considered as an epizootic disease with a single cause. Although the disease occurs mostly in feeder cattle during or after shipment from the West, chronic lung lesions of earlier orgin are often seen at necropsy; many of these are bacteriologically sterile. The disease is almost nonexistent in Toronto stockyards where no western cattle are received.

Pasteurella multocida and *Pasteurella hemolytica* are "the most notorious opportunists" but other organisms are found. Neither a virus nor a rickettsia had been demonstrated, but one of these may be a forerunner of the Pasteurella infection.

The spread of shipping fever to native cattle of any age, and under no apparent stress, has been observed on numerous occasions. Although the typical syndrome is occasionally seen in shipping fever, "there is no justification in using the term (hemorrhagic septicemia) as a synonym for shipping fever."

The good response to certain sulfonamide and streptomycin therapy would seem to indicate that bacterial infection is a dominant factor. When these agents fail, terramycin® given intramuscularly, daily for three or four days, is often effective.—*G. R. Carter in Canad. J. Comp. Med., Aug. 1956.*

Is Trembling in Pigs Due to a Virus?—Trembling in pigs was previously considered, in Sweden, to be hereditary. On three occasions after a new boar had been added to the breeding station, trembling occurred in litters from boars which had previously sired only healthy pigs. The condition disappeared when the new boar was removed and did not occur in the offspring when affected animals from the same litter, were mated.—*Vet. Bull., Aug., 1956.*

Toxoplasmosis in Swine.—When toxoplasmosis occurred in a herd of pigs in Norway, 6 from a litter of 11 died in a few days with signs of respiratory involvement. At necropsy, fibrinous pneumonia, necrotic lymphadenitis, ulcerous enteritis and focal hepatitis, nephritis, and splenitis were found. Toxoplasma, which produced typical infection in mice, were recovered from affected organs. Only weak serological reactions developed.—*Vet. Bull., Aug., 1956.*

A Pathogenic Pleuropneumonia-like Organism from a Calf

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SINCE IT IS frequently difficult to reproduce a disease with cultures of pleuropneumonia-like organisms (PPLO), the role of these organisms as a cause of disease is often in question. Nevertheless, PPLO which are capable of reproducing disease from pure culture in chickens,[1] swine,[2] and goats[3] in North America have been recovered.

It is the purpose of this report to describe a case of arthritis in a calf caused by a pathogenic PPLO and to report on experimental studies to date. In so far as we know, this is the first reported case of PPLO arthritis in the bovine species in the United States since the eradication of bovine pleuropneumonia.

REPORT OF CASE

Clinical Data.—On Oct. 4, 1955, a 2-month-old male Hereford calf was presented to the clinic with a history of weakness and stiffness for a week and a serous nasal discharge for two days. Clinical examination revealed a temperature of 104.8 F., hyperpnea, dyspnea, musical râles, and pleuritic friction sounds. The calf had diarrhea and its general condition was poor. The front fetlock, and knee, elbow, stifle, and hock joints all were enlarged and painful.

Synovial fluid was aspirated and revealed a count of 5,100 white blood cells and of 2,000 red blood cells per cubic millimeter. The differential count of white blood cells in the synovial fluid showed 91 per cent neutrophils and 9 per cent mononuclear cells. On two successive days, bacteriological cultures of joint fluid were negative. A clinical diagnosis of suppurative arthritis and pneumonia was made and the calf was given 1,300,000 units of penicillin and 1.75 Gm. of streptomycin, followed by 350,000 units of penicillin and 0.75 Gm. of streptomycin daily for three days. The temperature dropped to 103 F. and remained there, but there was no other sign of improvement. The calf was presented for necropsy on the fifth day.

Necropsy Findings.—The calf was killed by electrocution and examined immediately. The digestive tract was normal except for three tricholiths in the rumen. The kidneys contained many scattered friable, yellowish green infarcts about 5 mm. in diameter. They were slightly elevated above the surface of the kidney and roughly wedge-shaped when sectioned. The bronchi contained white froth and the right and left apical and cardial lobes of the lung showed focal areas of red consolidation.

The affected joints were enlarged, firm, and nonfluctuant and, when opened, large, yellowish plaques of fibrin were found adherent to the synovial membrane (fig. 1). The synovial fluid was not increased and was yellowish and viscid. The inner surface of the capsule was slightly reddened and showed minor villus formation. Nearly every diarthrodial joint was affected, the leg joints most severely. Cartilaginous erosion was not observed.

Microscopic Findings.—Blocks of tissue were fixed in 10 per cent formalin and stained with hematoxylin and eosin. Liver sections showed mild lipidosis but otherwise were normal. The spleen showed focal congestion and accumulations of macrophages in the red pulp.

Lung sections showed bronchioles filled with neutrophils and alveoli containing neutrophils and septal cells. Many of the bronchi contained macrophages in the lamina propria and outside the muscularis mucosae. The lung changes were essentially those of acute bronchopneumonia.

A section through a renal infarct showed necrotic parenchymal elements and disintegrating inflammatory cells scattered throughout a coarse, hyalinized stroma. The infarct was surrounded by a capsule of granulation tissue with an inner layer of macrophages.

Sections through capsules of the joints of the limbs showed replacement of the synovial layer by granulation tissue and edema of the fibrous layer (fig. 2). The inner layer of granulation tissue was in-

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This investigation was supported in part by a grant from the Abbott Laboratories, North Chicago, Ill.

The authors express their appreciation to Drs. H. E. Adler and D. R. Cordy for their cooperation in this study.

filtrated by macrophages and a few neutrophils. The cells lining the joint cavity were swollen, dark-staining, and showed loss of polarity. Overlying this membrane and extending into the joint cavity was a dense layer of fibrin with beginning stages of organization in the basilar portions. The edematous fibrous layer of the joint capsule showed patchy areas of lymphocytic infiltration. Because of the fibrinous nature of the arthritis, PPLO infection was suspected and material was submitted for culture.

Bacteriological Findings.—Joint fluid, kidney, liver, lung, spleen, and lymph nodes were cultured for two days on Difco PPLO agar supplemented with horse serum (10%) and yeast hydrolysate (1%). Tissues were also seeded into an enrichment broth[4] and, after 48 hours of incubation at 37 C., cultures were streaked on PPLO agar, incubated another 48 hours, and examined microscopically.

The organisms and their colonies were recognized as typical of the PPLO group. Colonies stained by two techniques[5,6] exhibited a typical dome-shape with a denser central portion. Giemsa stains of smears from broth culture demonstrated typical minute coccoid bodies. Several subcultures on mediums free of bacteriostatic substance were performed to eliminate the possibility of an "L" form of bacteria.[7]

The fermentation reactions[8] of the organism showed acid but not gas in sucrose, dextrin, dextrose, mannitol, galactose, and maltose. Levulose and lactose were not fermented. The PPLO hemolyzed bovine erthrocytes and liquefied inspissated horse serum.

Experimental Studies

Following the isolation of the PPLO from the joints, kidney, and spleen of the arthritic calf, the pathogenicity of the organism was tested in calves, sheep, pigs, mice, and chicken embryos.

Experiment 1.—Method of Inoculation.—The culture used for inoculation was prepared by seeding a large number of PPLO colonies directly into PPLO broth which contained 0.1 per cent agar and 500 units of penicillin per milliliter. After 24 hour's incubation at 37 C., the inoculum was checked for viability and contamination on agar plates free of any inhibitory substance.

This inoculum was given intravenously to 2 calves (A and B), 1 year old (5 ml. each) and to 2 lambs (2 ml. each). Adult mice were injected intraperitoneally with 0.25 ml. and 10, 7-day-old embryonating chicken eggs were injected with 0.2 ml. into the yolk sac. Daily temperatures were recorded in the calves and lambs. Blood samples were collected prior to inoculation to verify the absence of PPLO agglutinins in the inoculated animals. The calves and lambs were held for 20

Fig. 1—Hock joint from a calf with a field case of PPLO arthritis. The opened joint shows masses of fibrin attached to the reflected synovial membrane.

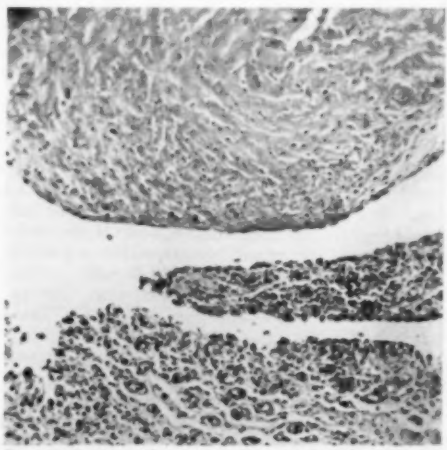

Fig. 2—Section through a synovial membrane from the calf with the natural infection of PPLO. The inflamed membrane (below) is covered with a layer of organized fibrin. There has been a separation of the membrane and the fibrin layer. x 300.

days and then killed for necropsy. The mice were killed after four weeks.

Clinical Findings.—The temperatures of the inoculated calves rose to 103 F. three days after injection and returned to normal three days later. During this period, the calves showed depression and anorexia. Seven days after inoculation, calf A exhibited sudden and severe lameness in the right hindleg but no swelling or warmth of the joints, and the temperature was not elevated. The calf showed increasing lameness and progressive inability to rise and walk. On the ninth day, it lay constantly and the right stifle was swollen and warm. On the twelfth day, when 5 ml. of PPLO broth was injected into the right stifle joint and then withdrawn, the resulting cultures for PPLO were negative. Until the twenty-first day, the calf showed continuous disinclination to rise and stand; however, after exercise its gait improved.

Calf B showed no elevation in temperature, but developed a progressive, general stiffness and a hobbling gait from the eighth day until the time of necropsy. At the same time, the conjunctiva of the right eye became swollen and reddened, and the cornea cloudy, then ulcerated. Two days following the development of the corneal lesion, a culture of ocular secretion was obtained by inserting a sterile cotton swab between the eyelids and suspending the collected material in PPLO broth. Pleuropneumonia-like organisms were recovered from this eye. The other eye was not cultured.

The temperatures and activity of the 2 lambs were not affected, but on the ninth day, both showed conjunctivitis. This resulted in an ulcerative keratitis in 1 lamb and a diffuse corneal cloudiness in the other.

White mice inoculated with the PPLO culture showed no disturbance during a four-week period of observation. Of 8 inoculated chicken embryos, 3 died after seven days.

Pathological Findings.—Necropsies of the experimental calves and sheep showed normal joints with no excess fluid and no inflammatory changes. Sections of joint capsule revealed no abnormalities.

Bacteriological Findings.—Joint fluid, kidney, liver, lung, spleen, and lymph nodes from the experimental calves and sheep were cultured in the same manner as in the clinical case. PPLO were recovered after a second passage on culture from the right stifle joint, liver, and spleen of calf A. Cultures from calf B and both lambs were negative after three such passages. No organisms were isolated from the mice. PPLO were present in large numbers in all inoculated eggs. The recovered PPLO showed the same staining characteristics, fermentation reactions, and liquefaction of inspissated horse serum as did the organisms isolated from the natural case.

Agglutination Test.—Slide-agglutination tests of the serums from the calves and lambs were conducted.[9] Preinoculation serums were all negative. The serums of calves A and B showed agglutination at dilutions of 1:16 and 1:4, respectively, at 13 days, but only at dilutions of 1:2 and 1:4, respectively, 20 days after inoculation. Undiluted sheep serums obtained at the time of necropsy agglutinated the antigen but were negative when diluted.

To determine the similarity between the injected and recovered PPLO in the experimental animals, antigen was prepared from the PPLO isolated in calf A. Serums obtained late from the calves and sheep agglutinated this antigen at the same dilution rate as the antigen prepared from the original inoculum.

Experiment 2.—The experiment was repeated using 2 steers (C and D), 18 months old, and 2 pigs, 4 months old. PPLO inoculum was injected directly into the stifle joint of steer C (5 ml.), intravenously in steer D (5 ml.), and into 2 pigs (2 ml.) by the intravenous and intraperitoneal routes, respectively. The inoculum, prepared as previously described, had been seeded in the field case PPLO strain which had been stored under refrigeration for two months.

None of the 4 animals exhibited any temperature elevation during 35 days of observation. Two days after inoculation, steer C developed swelling and warmth of the injected joint, with difficulty in rising, and lameness. This condition became increasingly severe for one week, then improved progressively but was still noticeable at necropsy. Steer D and the 2 pigs showed no particular involvement.

At necropsy, steer C had no conspicuous lesions. The injected stifle joint presented a normal amount of synovial fluid and no fibrin was observed. No abnormalities were found in the other animals.

PPLO cultures were made and organisms were recovered from the stifle joint of steer C. All other cultures were free of PPLO. Preinoculation serums were negative against PPLO antigens. The agglutination titer reached 1:64 and 1:4 in steers C and D, respectively, 16 days after inoculation. The pigs produced no agglutinins.

DISCUSSION

The ubiquity of the pleuropneumonia-like organisms permits speculation as to whether they are pathogenic or merely secondary to a previous infectious agent. In the case reported here, however, we seem to have demonstrated the pathogenicity of the isolated organism.

The lack of severity of the experimental infection is not surprising since reproduction of diseases caused by the PPLO group is generally difficult and requires special techniques. Nocard and Leclainche[10] demonstrated the organism in bovine pleuropneumonia in 1898, but they were unable to reproduce typical lung lesions until three years later. Not until 1938 was typical pleuropneumonia produced in many experimental animals (41%).[11] Our technique of inoculation may need improvement before complete results can be obtained.

Some PPLO pathogens lose their viru-

lence rapidly after several passages on artificial medium.[7] It, therefore, seems advisable to inject inflammatory exudate rather than culture in future experiments.

In the first experiment, despite the fact that lesions were absent at necropsy, marked lameness was produced in 1 calf and PPLO were recovered from the affected joint 20 days later.

The presence of the PPLO in the joints, kidneys, and spleen in the field case and in the joints, liver, and spleen in the experimental calf demonstrate the septicemic character of the disease. Of interest is the tendency of the organism to produce ocular lesions in both calves and lambs. The main lesions in the field case were located in the joints and this confirms a certain predilection of the PPLO group for articular tissue. Similar observations have been made in bovine pleuropneumonia, agalactia, chronic respiratory disease in chickens, rodent and human arthritis and, more recently, in arthritis in goats.

In the second experiment, the lack of pathogenicity may be attributed to a loss of virulence, probably due to refrigeration of the organism for two months, but the experimental steers differed from the field case by being older, and from the first experimental calves by their state of good nutrition. It is possible that animals are more susceptible when they are young or when their defense mechanisms are weakened by their poor nutritional condition.

The absence of reaction in the sheep, swine, and mice indicates a certain specificity of this PPLO strain for the bovine species.

The biochemical reactions of this organism resemble those of caprine and bovine pleuropneumonia organisms except that it ferments galactose. It differs from the strain isolated from goats[3] because it also ferments sucrose. No serological tests were performed to determine the relationship of this organism with those from the recognized PPLO diseases. However, it is our intention to test this organism against type serums of bovine pleuropneumonia and agalactia.

Summary

1) An organism was recovered from the joints, kidneys, and spleen of a calf showing severe arthritis and bronchopneumonia. Medication with penicillin and streptomycin had not been successful.

2) Culturally, the organism presented the characteristics of the PPLO group. It was not an "L" form of bacterium as demonstrated by subculturing on noninhibitory medium.

3) Biochemically, this organism seemed to be related to caprine and bovine pleuropneumonia organisms, except that it fermented galactose.

4) Experimental inoculation of young cattle caused clinical signs of stiffness, lameness, and keratitis. The 2 inoculated lambs developed keratisis. Agglutinins were demonstrated in the serums of these animals. Pigs and mice were unaffected. It was possible to make PPLO transmission through embryonating eggs.

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Electric shockers are used to temporarily paralyze fish while making stream population counts.—*Sci. News Letter, Aug. 18, 1956.*

Preliminary Studies on the Prophylactic and Therapeutic Values of Type B Vesicular Exanthema Immune Serum

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IT HAS BEEN shown[1] that the A type of vesicular exanthema immune serum has considerable prophylactic value in swine against homologous challenge.

This paper is a preliminary report of the therapeutic and prophylactic value of immune serum against the B type of vesicular exanthema virus.

MATERIALS AND METHODS

Preparation of Immune Serum.—The immune serum was prepared in swine by the inoculation of 1.0 ml. of a 1:10 dilution of tissue infected with B-type vesicular exanthema virus into 4 test animals. All inoculated animals responded with typical symptoms of vesicular exanthema. They were allowed to convalesce for 30 days, following which they were reinoculated with 1.0 ml. of a 1:10 dilution of the virus subcutaneously. No clinical evidence of the disease was noted after the second inoculation of virus. Ten days later, these animals were bled and their serums pooled and preserved with 1:50,000 merthiolate solution.

Challenge Virus.—The challenge virus consisted of a 1:10 dilution of the standard 10 per cent swine tissue suspension of type B vesicular exanthema virus. Unless otherwise indicated, all virus injections were made intradermally in the snout and upper lip.

EXPERIMENTAL PROCEDURE

Therapeutic Test.—A series of 20 pigs, weighing approximately 40 lb. each, were inoculated with 0.75 ml. of standard 10 per cent virus suspension. The pigs were then placed in three groups. Group 1 (7 pigs) received 20 ml. of serum subcutaneously into the axillary space four hours after inoculation with the virus. Group 2 (7 pigs) received an identical dose of serum 24 hours after inoculation with the virus. Group 3 (6 pigs) received no serum and served as untreated control pigs.

Prophylactic Test.—A second series of 20 similar pigs was inoculated subcutaneously in the right axillary space with 10 ml. of the immune serum. They were then separated into two groups of 10 pigs each. Group 1 was challenged at 14 days, and group 2 at 28 days, after receiving serum. Three untreated control animals were added to each group, making a total of 13 pigs per group.

Challenge of these two groups of the serum-treated pigs was identical; 3 received 0.75 ml. of virus while the remaining 7 served as contact exposed animals. The 3 control animals were also inoculated with 0.75 ml. of a 1:10 dilution of virus.

All animals of a group were kept in one pen, were observed regularly for ten days following challenge and intermittently for an additional 21 days, then all serum-treated pigs which had not shown clinical vesicular exanthema were challenged with live virus.

This second challenge consisted of 0.75 ml. of a 1:10 dilution of the B-type virus, also inoculated intradermally into the snout and upper lip. Three additional susceptible control pigs were included for each such second challenge group.

RESULTS

The results obtained from the therapeutic trials are shown (table 1). In group 1 (serum 4 hours after virus), 5 of 7 animals were protected against generalized vesicular exanthema; the other 2 appeared to be completely unprotected. In group 2 (serum 24 hours after virus), 3 of 7 animals were completely protected; 1 had lesions only at the site of inoculation, and 3 showed generalized vesicular exanthema. The 6 control pigs all responded with generalized vesicular exanthema. Of interest, although difficult to evaluate, were the febrile reactions of the animals. Temperatures above 105 F. were recorded in 4 of 6 control pigs and in only 1 of 14 serum-treated animals. Moreover, there was a considerable inflammatory response in all controls, but in only 4 of the 14 serum-treated pigs.

The results of the prophylactic value of B-type immune serum are shown (table 2). In group 1, the 3 control pigs developed typical generalized vesicular exanthema; of 3 serum-treated pigs challenged directly,

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Grateful acknowledgement is made to Paul Andriese for his valuable technical assistance.

This work was supported in part by a contract between the University of California, Department of Bacteriology, and the Office of Naval Research.

The opinions contained in this report are not to be construed as reflecting the views of the Navy Department or the Naval Service at large. (Article 1252, U. S. Navy Regulations, 1948.)

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TABLE 1—Clinical Response of Swine Treated with 20 ml. of Homologous Immune Serum, Four and 24 Hours After Intradermal Inoculation with 0.75 ml. of a 1:10 Dilution of Type B Vesicular Exanthema Virus*

Serum group designation	Test pig (No.)	Clinical evidence of vesicular exanthema
Group 1— Received 20 ml. serum 4 hours after virus inoculation	210	Generalized vesicular exanthema.
	211	No pyrexia, small vesicle at site of inoculation.
	212	No evidence of vesicular exanthema.
	213	No evidence of vesicular exanthema.
	214	Vesicles, site of inoculation; foot lesion.
	215	No pyrexia, small vesicle at site of inoculation.
	216	No evidence of vesicular exanthema.
Control group— no serum	224	Severe, generalized vesicular exanthema.
	225	Severe, generalized vesicular exanthema.
	226	Severe, generalized vesicular exanthema.
	227	Severe, generalized vesicular exanthema.
	228	Severe, generalized vesicular exanthema.
	229	Severe, generalized vesicular exanthema.
Group 2— received 20 ml. serum 24 hours after virus inoculation	217	No evidence of vesicular exanthema.
	218	Pyrexia, snout lesions only.
	219	No evidence of vesicular exanthema.
	220	Pyrexia, generalized vesicular exanthema.
	221	Pyrexia, generalized vesicular exanthema.
	222	Pyrexia, generalized vesicular exanthema.
	223	No evidence of vesicular exanthema.

*Challenge virus consisted of a 1:10 dilution of the 10 per cent tissue suspension of type B vesicular exanthema virus.

TABLE 2—Clinical Response of Swine Inoculated with 10 ml. of Homologous Immune Serum 14 and 28 Days Prior to Challenge* with Type B Vesicular Exanthema Virus

Serum group designation	Test pig (No.)	Challenge schedule		Clinical response
		Route	Amount	
Group 1— received 10 ml. serum 14 days prior to challenge	187	i.d.†	0.75 ml.	Minute lesion, inoculation site.
	193	i.d.	0.75 ml.	None.
	196	i.d.	0.75 ml.	Small tongue lesion only.
	188	C**	Unknown	None.
	189	C	Unknown	None.
	190	C	Unknown	None.
	191	C	Unknown	None.
	192	C	Unknown	None.
	194	C	Unknown	None.
	195	C	Unknown	None.
Control group— no serum	200	i.d.	0.75 ml.	Generalized vesicular exanthema.
	203	i.d.	0.75 ml.	Generalized vesicular exanthema.
	209	i.d.	0.75 ml.	Generalized vesicular exanthema.
Group 2— received 10 ml. serum 28 days prior to challenge	183	i.d.	0.75 ml.	Generalized vesicular exanthema.
	184	i.d.	0.75 ml.	Snout lesions only.
	198	i.d.	0.75 ml.	Generalized vesicular exanthema.
	177	C	Unknown	None.
	178	C	Unknown	None.
	181	C	Unknown	None.
	182	C	Unknown	Snout lesions only.
	185	C	Unknown	Generalized vesicular exanthema.
	186	C	Unknown	Generalized vesicular exanthema.
	199	C	Unknown	None.
Control group— no serum	197	i.d.	0.75 ml.	Generalized vesicular exanthema.
	201	i.d.	0.75 ml.	Generalized vesicular exanthema.
	202	i.d.	0.75 ml.	Generalized vesicular exanthema.

*Challenge virus consisted of a 1:10 dilution of the standard 10 per cent tissue suspension of type B vesicular exanthema virus.

†i.d. = intradermal inoculation into snout and upper lip.

**C = contact exposure only.

1 showed no lesions while 2 showed only minute lesions; and of the 8 challenged by contact, 3 showed no reaction, while 5 showed only a mild rise in temperature.

In group 2, the control animals again developed typical vesicular exanthema, while the serum-treated animals showed some evidence of protection. Four, all challenged by contact, had no evidence of the disease; 2 developed snout lesions only; and 4 developed generalized vesicular exanthema. These results compare favorably with those reported[1] for the A-type immune serum.

Approximately 30 to 35 days after they were challenged either directly or by contact, all animals not showing clinical signs of vesicular exanthema showed solid immunity when rechallenged with B virus. This result was identical to that reported[1] for the A-type immune serum.

It appears reasonable to conclude that animals receiving immune serum, followed by exposure to the virus, whether by direct inoculation or contact with infected animals, acquire solid immunity against the disease, despite the absence of clinical symptoms.

DISCUSSION AND CONCLUSIONS

The therapeutic trials with vesicular exanthema immune serum indicate that immune serum would be of considerable value in the treatment of exposed swine of all ages, providing the serum was administered during the first 24 hours after exposure.

The results of the prophylactic trials were almost identical with those previously reported for the A-type immune serum.[1] The B-type serum was effective for as long as 14 days in either completely protecting swine or in preventing the generalized response and reducing the severity of the localized lesion. Twenty-eight days after inoculation with serum, 40 per cent responded like fully susceptible swine. The remaining 60 per cent were either completely protected or the generalized response was prevented. Animals which had received serum, followed by challenge, were then actively immune even in the absence of clinical symptoms.

The addition of an effective immune serum to the armamentarium of the veterinarian against this vesicular disease has been discussed by Madin and Traum[2] and should be another step forward in the control and management of the disease. It is believed that such an immune serum could be made readily available with sufficient research and interest.

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Bovine Ketosis in Australia

In a four-year period, 152 typical cases of bovine ketosis were reported from one practice. It seemed most common in less fertile areas and during periods when the dietary intake was poor. There seemed to be a relationship between its prevalence and rainfall, with an increase when precipitation was low.—*Austral. Vet. J., Feb., 1956.*

Leptospiral Meningitis in Cattle.—A disease of cattle marked by sudden onset, incoordination, and followed by death has been common in cattle in Turkey. Jaundice and fever are absent, the principal lesion being serous meningitis. Leptospira were observed in smears of the meninges and *Leptospira pomona* was isolated from some lesions.—*Vet. Bull., July, 1956.*

Phenothiazine and Carob Flour for Bovine Stomach Worms.—Phenothiazine plus carob flour (3 parts to 1), when mixed with the feed at the rate of 1.5 oz. per animal per day for two days, was effective against stomach worms in cattle. The flour masked the undesirable flavor of the phenothiazine and prevented its absorption, thus increasing the margin of safety.—*California Vet., Sept.-Oct., 1955.*

Allergic reactions in cattle vaccinated against foot-and-mouth disease have increased, in Germany, from negligible prior to 1952, to between 3 and 10 per cent since then. It is suggested that nonspecific antigens in the vaccine may act as allergens.—*Vet. Bull., July, 1956.*

A virus was isolated from turkeys with chronic respiratory disease. It was resistant to antibiotics and not neutralized by immune serums for Newcastle disease, infectious bronchitis, and infectious laryngotracheitis.—*Vet. Bull., July, 1956.*

What Is Your Diagnosis?

Because of the interest in veterinary radiology, a case history and accompanying radiographs depicting a diagnostic problem are usually published in each issue of the JOURNAL.

Make your diagnosis from the picture below — then turn the page ▸

History.—A Hereford bull, 1 year old, in show condition and negative to tests for brucellosis and tuberculosis, was brought from his stall to serve a cow. He made one attempt to mount, then could not be persuaded to try again. The following day he moved with difficulty. Posterior paralysis developed gradually and in one week he could no longer rise. Death occurred two days later. At necropsy, one region was quite hemorrhagic and inflamed. This section was removed and the radiograph shown here was taken.

(Diagnosis and findings are reported on the next page)

Here Is the Diagnosis

(*Continued from preceding page*)

Diagnosis.—Degeneration of the lumbar vertebrae as the result of osteomyelitis.

Comments.—Paralysis was due to injury of the spinal cord by pressure from the degenerating vertebrae. To ascertain that the osteomyelitis was not the result of Brucella infection, cultures were made from these tissues. They were negative.

Our readers are invited to submit case histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

This case was submitted by the clinical staff of the School of Veterinary Medicine, University of Illinois, Urbana.

Some Effects of Chlorpromazine Hydrochloride in Horses

Chlorpromazine hydrochloride (thorazine®) was employed in 28 experiments on 6 horses; the effect on heart rate, breathing rate and pattern, blood pressure, body temperature, and behavior was studied. Doses of 2.0 to 4.0 mg. per kilogram of body weight, intramuscularly, produced a slight tachycardia and hypotension; a slower, more regular and deeper breathing pattern; and a slight fall in body temperature. Doses of 2.5 mg./kg. or larger resulted in signs of moderate-to-marked depression within one hour, lasting at least six hours and, in some instances, for 24 to 48 hours.

The animals employed were confined under quiet conditions; larger doses of the drug may be necessary to depress excitable horses. In all instances, a 5 per cent solution of the drug was employed. This may result in slight, local irritation.

Chlorpromazine hydrochloride in doses of 1.5 or 2.0 mg./kg., combined with meperidine hydrochloride (demerol®)* in doses of 750.0 or 500.0 mg., respectively, produced a degree of depression comparable to that seen with larger doses of chlorpromazine alone.

Repeated doses of chlorpromazine hydrochloride over a three-day (2 mg./kg. daily) or five-day (1 mg./kg. daily) period resulted in a fall in circulating red blood cells and in hemoglobin concentration, sometimes for at least two weeks. Further studies on this activity are needed.

When a mare, which had developed a belligerent attitude toward her foal and refused to allow it to nurse, was injected with chlorpromazine (1 mg./kg.), she allowed the foal to nurse in six hours, the first time in four days. Following this, she showed a normal brood mare type of behavior.—[*John E. Martin and John D. Beck: Some Effects of Chlorpromazine Hydrochloride in Horses. Am. J. Vet. Res., 17, (Oct., 1956): 678-686.*]

*The meperidine hydrochloride (demerol) employed in these studies was supplied by Winthrop-Stearns Co., New York, N. Y.

Antibiotics Hamper Diagnosis

Bacterial cultures were inhibited and blood smears were negative from a cow which was slaughtered, in Holland, after being treated for two days with penicillin and streptomycin. However, organisms resembling *Bacillus anthracis* were seen in smears of the cultures. Two mice inoculated with the culture died in two and five days and typical anthrax bacilli were found in spleen smears of each. The authors requested practitioners to notify the meat inspection service of animals that had been treated with antibiotics before slaughter. —*Vet. Bull., Aug. 1956.*

[A similar difficulty has occurred for years with regard to swine erysipelas. Laboratory diagnosticians have requested that specimens submitted be from swine which had not been treated with penicillin.—ED.]

The Absorption of Chlortetracycline (Aureomycin) by the Bovine Uterus

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ANTIBIOTICS are routinely introduced into the bovine reproductive tract to aid in the control of bacterial infections. Although the use of these agents is intended, almost without exception, for local action on the endometrium, a previous study[1] has shown that sulfamethazine introduced into the uterus is quickly absorbed and that therapeutic blood levels result. These results suggest that the absorption of antibiotics by the bovine uterus should also be investigated. Chlortetracycline (aureomycin®*), used frequently in the treatment of the bovine endometrium, was selected for this purpose.

Although there have been no controlled studies on the absorption of antibiotics by the bovine uterus, the absorption of aureomycin from the bovine mammary gland has been investigated. After infusion of the mammary gland with 400 mg. of aureomycin (in ointment form) per quarter, a peak blood serum concentration varying from 0.1 to 0.8 μg. per milliliter was reached in 12 hours.[2] In another study,[3] after aureomycin was infused in amounts up to 1.0 Gm., blood serum levels were less than 1.0 μg. per milliliter; and when 2.5 Gm. was given intravenously, the serum levels ranged from 1.0 to 12.4 μg. per milliliter, with peak concentrations reached in the first hour.

PROCEDURE

Ten cows, which had completed at least one gestation, were studied. They were nonpregnant, nonlactating, and were in the diestrous phase of the estrous cycle, as determined by estrous cycle history and confirmed by rectal palpation. Each cow was given 40 cc. of aqueous solution containing 1 Gm. of crystalline aureomycin. In 8 cows, the solution was deposited in the lumen of the uterus with a Chamber's catheter and in 2 cows it was injected intravenously. Jugular blood samples were taken before injections and at ½-, 1-, 2-, 4-, 8-, 12-, and 24-hour intervals after administration of the drug. The samples were allowed to clot and the serum was withdrawn and frozen as soon as possible. The samples were assayed for antibiotic activity,† using the Food and Drug Administration agar-diffusion method.[4]

RESULTS AND DISCUSSION

The rate of absorption from the uterus was uniform, with the peak concentrations usually reached in two hours (table 1). This is similar to the absorption rate of sulfamethazine by the bovine uterus and, although the process has not been extensively studied, it may indicate a rapid absorption rate for all aqueous solutions introduced into the uterus. The blood serum levels varied considerably (0.053 to 0.18 μg./ml. at peak) in individual animals. This same observation was made by others[2] during their investigation of mammary ab-

TABLE 1—Aureomycin Content of Serum from Cows Infused in Lumen of the Uterus with 1 Gm. of Aureomycin* (μg./ml.)

Hours	Animals								Mean
	222	226	324	534	566	429	630	755	
0	0.025	0.025	0.01	0.01	0.01	0.01	0.025	0.025	0.018
½	0.10	0.028	0.032	0.029	0.11	0.03	0.045	0.053	0.053
1	0.12	0.088	0.038	0.045	0.112	0.037	0.066	0.073	0.072
2	0.18	0.11	0.053	0.067	0.098	0.053	0.064	0.072	0.087
4	0.088	0.076	0.039	0.085	0.094	0.051	0.068	0.074	0.072
8	0.049	0.061	0.038	0.045	0.089	0.041	0.062	0.072	0.057
12	0.039	0.047	0.021	0.033	0.037	0.026	0.047	0.072	0.043
24	0.034	0.028	0.015	0.039	0.033	0.025	0.050	0.051	0.037

*Aureomycin, crystalline 1 Gm.

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Approved by the director of the Missouri Agricultural Experiment Station, Columbia; journal series No. 1603.

*Aureomycin supplied by Lederle Laboratories Division, American Cyanamid Co., Pearl River, N. Y.

†The authors express thanks to A. C. Dornbush, Ph.D., personally, and the Research Division, American Cyanamid Co., Pearl River, N. Y., for assay of the blood serum.

TABLE 2—Aureomycin Content of Serum from Cows Injected Intravenously with 1 Gm. of Aureomycin* (μg./ml.)

Hours	Animals 128	175	Mean
0	0.025	0.025	0.025
½	1.5	1.5	1.5
1	1.0	1.4	1.2
2	0.76	0.63	0.69
4	0.49	0.82	0.65
8	0.28	0.27	0.27
12	0.097	0.12	0.10
24	0.034	0.032	0.033

*Aureomycin, crystalline 1 Gm.

sorption of aureomycin. The relatively low serum levels of aureomycin activity following intrauterine administration may have been due, in part, to the way the samples were handled following collection. Aureomycin has been found unstable[4] in the presence of serum or other body fluids when held at room temperature for relatively short periods, and storage of samples in a common electric refrigerator has resulted in complete loss of aureomycin activity in some samples. However, as noted in the procedure, blood samples of both the intrauterine and intravenous studies were handled in the same way during this investigation.

The blood serum levels following intravenous administration (table 2) were highest 30 minutes after injection (1.5 μg./ml.), then receded until similar to the levels following intrauterine injections at 24 hours. They were within the range found following higher intravenous dosages of the drug.[3]

Another cause of variation in blood concentrations of the drug may have been the reaction of the myometrium to the intrauterine injection; this varied from intense contraction of the uterus in a few cows to little or no reaction in others. Strong myometrial contractions expelled some of the solution from the uterus as evidenced by vaginal discharge.

Urine samples were not taken, for it has been shown[5] that only 15 to 20 per cent of the antibiotic is recovered in urine even after intravenous administration.

SUMMARY

1) Aureomycin® is rapidly absorbed into the blood stream after infusion into the lumen of the uterus.

2) The peak blood serum level of aureomycin activity is reached approximately two hours after intrauterine infusion.

3) A higher level of aureomycin activity is found in the blood serum for the first 24 hours following intravenous administration than is attained following intrauterine administration.

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Effect of Antibiotics on Intestines.—The average weight of the intestinal tract of chickens fed antibiotics was less than that of controls; the differences were often significant. This was not due to a shortening of the tract or to a reduction in fat or moisture content. The duodenum was usually of smaller diameter, with a thinner tunica propria and shorter villi. The mesenteric blood vessels were not affected.—*Poult. Sci., May, 1956.*

How Chlorpromazine Causes Jaundice.—Pathological changes in the liver following the use of chlorpromazine were studied in specimens obtained by biopsy or autopsy from four persons. The intrahepatic canalicular obstruction suggested that chlorpromazine produced an increased viscosity of the bile, leading to stasis in the smaller bile passages.—*J. Am.M.A., June 9, 1956.*

Placebo pills had the same effect as reserpine in relieving anxiety, at Northwestern University, when fed to patients for six days before surgery.—*Science, July 27, 1956.*

Clostridium Perfringens Type D *(Epsilon)* Enterotoxemia in Brown Swiss Dairy Calves

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THE PURPOSE of this paper is to report the occurrence of *Clostridium perfringens* (*welchii*) type D (*epsilon*) toxin in the intestines of dairy calves dying from acute enterotoxemia in Weld County, Colorado.

LITERATURE CITED

The cause of enterotoxemia was first established in 1932[1] in western Australia when the presence of *Cl. perfringens* type D toxins in the small intestines of sheep was demonstrated. Since then considerable bibliography on *Cl. perfringens* type D in sheep has been compiled. The literature contains few reports of *Cl. perfringens* enterotoxemia in cattle. In 1936, type A toxin was recovered from calves in Australia.[2] An enterotoxemia in calves caused by a type of *Cl. perfringens* that has been designated type E was reported in 1936.[3] An enterotoxemia was described in young suckling calves similar to enterotoxemia in lambs from which *Cl. perfringens* type A organisms[4] were recovered. In 1952, a case of necrotic enterotoxemia in a calf due to *Cl. perfringens* type B was reported.[5] In 1953, a hemorrhagic enterotoxemia in young suckling calves was described.[6] *Clostridium perfringens* type C toxins and organisms were recovered from the intestinal contents. From Australia, in 1954, the recovery of *Cl. perfringens* type D toxin from the intestines of adult cattle dead from enterotoxemia was reported.[7]

HERD HISTORY

Three calves 7, 8, and 10 weeks old, in a herd of 65 Brown Swiss, had died suddenly on a dairy farm in Weld County, Colorado. The environmental conditions and state of nutrition of the calves was good. They were held in individual calf pens in a large horse barn which was, at times, drafty. The calves were fed alfalfa hay free choice and a grain concentrate calf starter. One gallon of skim milk with added vitamin A was bucket-fed at each of two daily feedings. Seven calves were less than 8 months of age.

CASE REPORTS

Case 1.—A heifer calf, 8 weeks old, was reported as acutely ill Feb. 26, 1956, at 1:30 p.m. The attending veterinarians* found her in a moribund condition, with a temperature of 95 F. She was given 20 cc. each of *Cl. perfringens* type C and type D antitoxin on the morning of February 27 and died that evening. A necropsy was performed on the premises.

Case 2.—On March 28, a 7-week-old heifer calf which had consumed her morning feed and had appeared normal at 11:30 a.m., was found down in the evening. She was dead on arrival of the veterinarian at 7:15 p.m. The next morning, the calf was brought to the veterinary hospital, Colorado A. & M. College, for necropsy and laboratory studies.

Case 3.—A third heifer, 10 weeks old, was found down at 8:00 a.m. on April 4. She was dead on the arrival of the veterinarian at 8:30 a.m. She had been treated on January 27 for pneumonia and had shown satisfactory evidence of recovery. Shortly after death, this calf was also brought to the hospital for studies.

DISCUSSION

Each of the 3 calves had appeared normal at the time of their last feeding. The first sign of illness in each was prostration in lateral recumbency. All appeared colicky and kicked spasmodically. No bawling was heard, but they grunted frequently. Each had a subnormal temperature, sunken eyes, and the head was held in a position of opisthotonos. There was no evidence of diarrhea. Clinically, the calves presented a typical picture of enterotoxemia as seen in feeder lambs. Two calves died within four or five hours after being found sick; 1 lived for approximately 36 hours.

At necropsy, very few pathological changes were recognized. The small intestines had areas of mucosal hyperemia, and the ileal segment particularly presented evidence of a mild serocatarrhal enteritis. The intestinal contents were watery and yellowish and, when centrifuged, the supernatant fluid was light red. The livers were mildly swollen. A few subepicardial hemorrhages were observed. The mesenteric nodes were serohemorrhagic. All other

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Published with the permission of the director, Colorado Agricultural Experiment Station as scientific journal paper No. 482.

*Drs. W. W. Aichelman and G. D. Brown, Fort Lupton, Colo.

organs and tissues appeared normal except that the cardiac and apical lobes of the lung from the third calf were red and consolidated, with evidence of resolution in the form of small foci of inflated normal pulmonary tissue.

Smears of the intestinal contents from the second and third calves revealed tissue debris and numerous bacteria, the majority of which were gram-positive rods morphologically resembling *Cl. perfringens*. Bacteria-free supernatant fluid from the intestinal contents were lethal to white mice. All mice inoculated with 0.1 cc. and 0.2 cc. died within 20 minutes. Identification by neutralization of the toxin was achieved by adding 0.3 cc. of types A, B, C, or D antitoxin, respectively, to four tubes containing 1.0 cc. each of intestinal supernatant fluid and incubating the mixtures at room temperature for 45 minutes. Each of the above described mixtures was inoculated (0.3 cc.) into 2 test mice. The toxic constituent of the intestinal content was neutralized by types B and D antitoxin. All mice inoculated with mixtures of types A and C antitoxin and intestinal content died within a few minutes. The neutralization test indicated that the toxic substance was the *epsilon* toxin produced by types D and B *Cl. perfringens*. Cultures of *Cl. perfringens* recovered from the intestinal contents produced *epsilon* toxin which is the primary toxic fraction of the type D organism.

SUMMARY

Dairy calves died suddenly of enterotoxemia. Diagnosis was based on recovery of *Clostridium perfringens* type D toxins and organisms from intestinal contents.

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The Use of Magnets in the Control of Traumatic Gastritis of Cattle

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In October, 1955, a preliminary report[1] was made on the efficacy of alnico bar magnets placed in the stomach of cattle for the control of bovine traumatic gastritis. Since the original group of 42 test heifers have now carried magnets for a year and a half, a more complete report may be made.

In the first six months of investigation, 2 of the original 42 heifers given magnets required rumenotomies.[1] In the succeeding ten months, only 1 additional heifer required surgical intervention. In her case, no metal could be found in the stomach other than that adherent to the magnet, but the foreign objects were of such a nature and, as a unit, so heavy that they caused ulceration of the floor of the reticulum. After removal of the offending metal and replacement of the magnet, the heifer made a satisfactory recovery.

Over 2,200 first- and second-calf heifers have been given magnets in the last six to 18 months within my practice area, 75 per cent of them in the past 12 months. To date, 22 of these animals have required surgical intervention to alleviate symptoms of traumatic gastritis. The following is a brief review of the findings in these 22 rumenotomy cases.*

In the majority of animals, it was found that the injury was not caused by wire penetration but, rather, by wire irritation. This is exemplified by a heifer in which a small wire on the end of the magnet had caused reticular irritation. The magnet was cleaned and replaced and the heifer was giving 70 lb. of milk a day four days after surgery.

In other instances, the wire was longer

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*Drs. R. V. Jessup and C. B. Nelson, veterinarians at the Roger Jessup Farms, Glendale, Calif., assisted with these cases.

[1]Carroll, R. E.: Magnets in the Control of Traumatic Gastritis. J.A.V.M.A., 127, (1955): 311.

Fig. 1 (Insert)—Magnet recovered at necropsy from yearling heifer after being in the stomach three weeks. The same magnet is shown in the larger picture along with the metal objects which were separated from it.

than the magnet and the projecting end of the wire was penetrating the wall of the reticulum. (To reduce this possibility, the length of the magnets has been increased from 2½ to 3 inches.) In one case, a 5-inch piece of baling wire attached to the magnet was found penetrating the "honeycomb" mucosal folds of the reticulum. This magnet had 19 assorted pieces of metal attached to it.

In 2 heifers on which surgery was performed it was evident, by the nature and type of the offending wire, that the penetrating foreign body had been in the animal longer than the magnet. In these instances, the wire may have been in the act of penetration when the magnet was administered. In another instance, the magnet had been demagnetized.

Of interest are the findings in a heifer destroyed because of signs of tetanus. She was one of a large group of yearling dairy heifers which had been given magnets. The heifers were being raised as replacement stock on a premise where the "hardware" problem was acute, probably due to the carelessly handled cheap feed (carrots, parsnips, greens, etc.) which was being fed to the lactating and dry cows as well as to the replacement heifers. A negligible amount of grain and alfalfa hay was also being fed. Shortly after dehorning and three weeks after magnet administration, this heifer developed symptoms of tetanus. At necropsy, when the magnet was recovered from the floor of the reticulum, it was covered with nails, wires, staples, iron filings, etc. (fig. 1). A hand magnet was passed over the interior walls of both rumen and reticulum, but no foreign material other than that attached to the magnet was found.

CONCLUSION

The frequence of traumatic gastritis in cattle was reduced over previous years, apparently by the administration of alnico bar magnets. However, the 1 per cent occurrence, as represented by 22 rumenotomies required in 2,200 magnet-treated animals, probably is lower than should usually be expected. In one herd where 70 per cent of the adult cattle were given magnets, the frequency of signs of traumatic gastritis in a ten-month period, July, 1955, through April, 1956, was reduced by 50 per cent as compared with a like period in 1954-1955. The prevalence of traumatic gastritis symptoms in animals without magnets was approximately the same in both years.

The increase in magnet length from 2½ inches to 3 inches should result in greater efficiency by reducing the overlap of longer pieces of wire. The benefit from using magnets in prophylaxis rather than in therapy was demonstrated. Thus, magnets should

be most effective when given to first- or second-calf heifers, since there is a minimum of foreign body injury in that age group.

The Longitudinal Vertebral Venous Sinuses of the Dog—An Abstract

Recent studies of the venous sinuses of the spinal canal have suggested a solution for many perplexing questions of metastasis and collateral circulation in clinical medicine in man. Part I records and illustrates the anatomy of the longitudinal vertebral venous sinuses from the dissection of 47 dogs. The vessels are paired and valveless and extend along the floor of the epidural space from the venous sinuses of the cranium. They are much less plexiform than those in man. The sinuses are vulnerable during clinical procedures which invade the atlanto-occiptal and the lumbosacral spaces.

Part II of the article explores the functional aspects of the sinuses in 22 experimental dogs. Venographs, following diodrast® injection, chart the route of venous return after a number of large veins were ligated. Numerous attempts, without marked results, were made to influence venous flow by increasing body pressures.

The vertebral sinuses are the largest and most immediately available of the collateral routes of venous return after ligation of the abdominal vena cava or both external jugular veins. They are capable of enlarging their caliber, as are their tributary and emissary radicles. Following such ligations, no serious effects were seen during postoperative observation periods up to six months.

The presence and competency of their valves and the variable amount of thrombus formation influence the number and combinations of other collateral pathways which enlarge after ligation. Most of these were previously existing, small anastomoses. Ligation of a number of them after previous caval occlusion does not embarrass venous return via the remaining routes. Alternate channels of venous flow are inadequate, however, when the intrathoracic posterior vena cava is suddenly occluded. Both external iliac veins were ligated without stasis of blood in the hindlimbs.

The clinical significance of the vertebral sinuses as a possible pathway for tumor

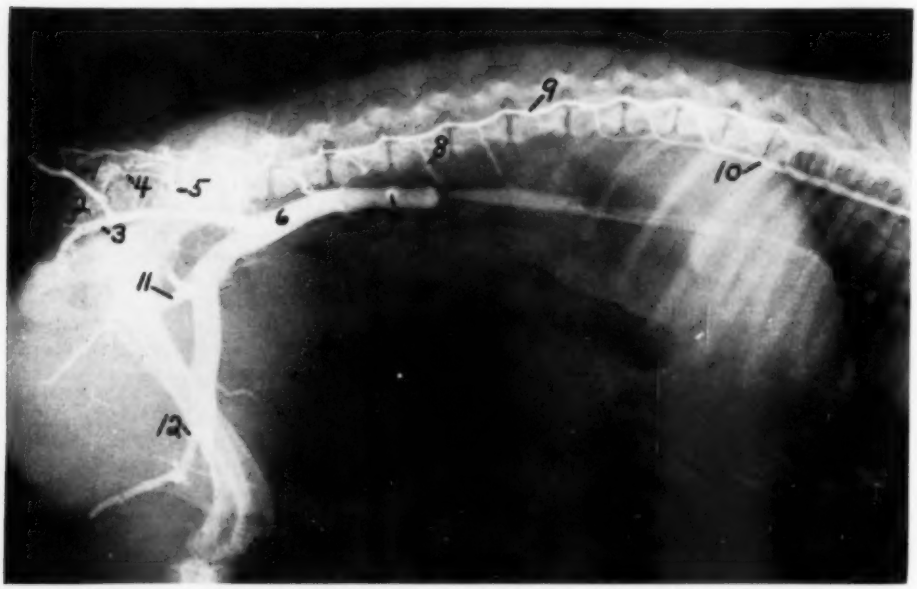

Fig. 1—Venograph of dog completed 45 minutes after ligation of the posterior vena cava. 1—Injection into right tail vein (v.); 2—posterior hemorrhoidal v.; 3—internal pudenal v.; 4—middle sacral v.; 5—anterior gluteal v.; 6—common iliac veins; 7—deep circumflex iliac v.; 8—fourth lumbar v. draining into posterior vena cava anterior to ligature (arrow); 9—longitudinal vertebral sinus; 10—azygos v.; 11—deep femoral veins; 12—valve in femoral v.

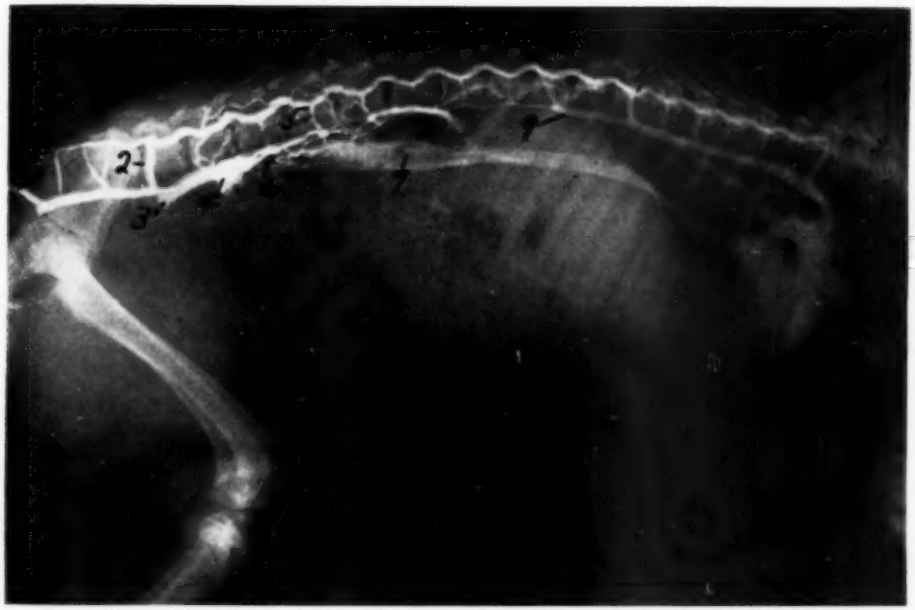

Fig. 2—Venograph of dog made six weeks after posterior vena cava ligation. 1—Injection into right tail vein (v.); 2—iliolumbar v.; 3—external iliac v.; 4—deep circumflex iliac v.; 5—basivertebral v.; 6—point of caval ligation; 7—entrance of renal v.; 8—phrenico-abdominal v.; 9—azygos v.; 10—longitudinal vertebral sinus.

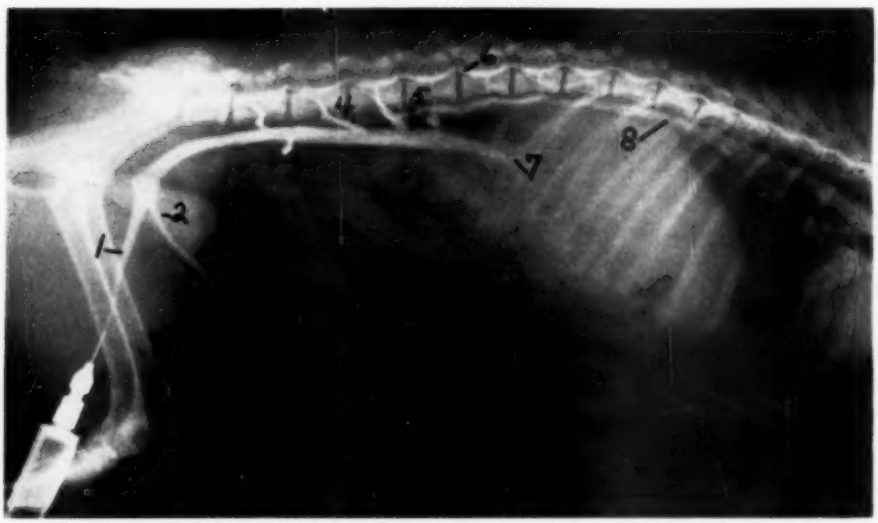

Fig. 3—Venograph of dog made 18 days after caval ligation anterior to the renal veins. 1—Injection into left femoral vein (v.); 2—posterior deep epigastric v.; 3—deep circumflex iliac v.; 4—fifth lumbar v.; 5—anastomosis between fourth and third lumbar veins; 6—longitudinal vertebral sinus; 7—point of caval ligation; 8—azygos v.

metastasis awaits further study since, contrary to the results reported in man and monkey, elevation of the intra-abdominal pressure did not appear to shift an appreciable amount of caval blood flow into the sinuses of the dogs used in this investigation.—[*R. P. Worthman: The Longitudinal Vertebral Venous Sinuses of the Dog. I. Anatomy. II. Functional Aspects. Am. J. Vet. Res., 17, (July, 1956) :341-363*]

An Epidemic of Q Fever in Algeria

Most of the men in two companies of French soldiers, recently arrived in Algeria, developed a griplike syndrome with fever, headache, and body pains. Soldiers in adjacent companies were not affected. Those affected had traveled in boxcars formerly used to transport animals, and they were quartered in a stable. Most of 20 patients tested were serologically positive to *Coxiella burnetti*. Most of the cases were brief and benign but 6 had a peculiar prolonged form for about a month; a few of the patients had relapses.—*J.Am.M.A., June 23, 1956.*

Effect of Disease on Parasites.—Several bacterial or viral infections apparently reduced the intensity of *Oesophagostomum dentatum* infection in swine. The number of pigs free of this parasite was highest in those which had swine erysipelas (91%), next in those with hog cholera (87%), and lowest in healthy pigs (37%). A high degree of infection was more common in healthy pigs (9.7%) than in those with cholera (3.6%), while none with swine erysipelas were severely infected.—*Vet. Bull., Aug., 1956.*

Insects commonly serve as vectors for plant viruses. Some insects transmit the virus directly to the next generation without an intermediate plant host. Others become infective for life, but only after an incubative period of several days.—*Science, Aug. 10, 1956.*

Adenoviruses—A Respiratory Group.—A committee has proposed the term, "adenovirus group" for viruses which produce acute infection of respiratory and ocular mucous membranes, with associated enlargement of lymphadenoid tissues, in the area and of the regional lymph glands. Of such viruses, 12 have been reported from man and 2 from monkeys. This designation is proposed until a satisfactory nomenclature can be established.—*Science, July 20, 1956.*

Lifesaving Antibacterial Agents

Largely because of the effect of antibacterial agents, the case fatality rates of human diseases of 20 years ago have dropped as follows: lobar pneumonia, from 25 to 4 per cent; pneumococcic meningitis, from 90 to 30 per cent; meningococcic meningitis, from 33 to 4 per cent; subacute bacterial endocarditis, from 100 to about 20 per cent; typhoid fever, from about 10 to 2 per cent; Rocky Mountain spotted fever, from 20 to about 1 per cent; and bubonic plague, from about 50 to 4 per cent.—*Am. J. Pub. Health, August, 1956.*

Interpreting Laboratory Reports

The practicing veterinarian alone is in a position to evaluate a report from a diagnostic laboratory. *Culturing the erysipelas bacillus from a pig could be misleading since pigs are often asymptomatic carriers of the disease.* Likewise, finding Leptospira in fish may not indicate the cause of the illness under consideration. Finding agglutinins against Leptospira in the serum is even more misleading. A laboratory report should always be weighed against conditions which pertain to the case in making the diagnosis.—*Georgia Vet., Oct., 1955.*

Penicillin Levels in the Injured.—Blood penicillin levels, six hours after the injection of 300 to 600 thousand units of aqueous procaine penicillin in 65 severely injured Korean war casualties, were below those of control subjects but well above the recommended minimum (0.03 to 0.06 units/ml.) for most penicillin-sensitive infections. However, the highest levels were not adequate for inhibition of many clostridia isolated from these wounds.—*Antibiot. Med. and Clin. Therap., July, 1956.*

Negri bodies were found in 3 pet skunks sold by a fur farm in Minnesota, although no animals on the farm had shown illness. Ten exposed persons were receiving antirabies vaccine.—*Pub. Health Serv., July 12, 1956.*

Isolation of the Dermatophyte, *Microsporum Gypseum*, from a Horse with Ringworm

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Chamblee, Georgia, and Columbus, Ohio

THE DERMATOPHYTE, *Microsporum gypseum (Microsporum fulvum, Achorion gypseum)*, has been shown[1,2] to be a relatively common, soil-inhabiting fungus. In one study, employing the "hair bait" technique,[3] isolations of *M. gypseum* were made from 37 (31.9%) of 116 soil samples collected from various locations in Tennessee and Georgia. Subsequent analyses of foreign and domestic soils[4] have resulted in equally frequent recoveries of this organism.

In view of the abundance of this fungus in nature and the apparent ample opportunity of exposure to this pathogen, it is surprising to find recorded in the world literature only 50 cases of *M. gypseum*-induced ringworm in horses.[5-13] All of these cases have been reported from Europe with the exception of 2 from Madagascar. One report[5] describes an outbreak of disease, in a Belgian remount station, involving 40 horses. Thus, it appears that *M. gypseum* is capable of producing epizootics of ringworm in horses. The status of this disease in horses in the United States is not known.

This report describes the clinical and mycological findings on a horse found to be spontaneously infected with *M. gypseum*, the first such authenticated equine infection to be reported from this country.

CASE HISTORY

The horse in question, a 20-month-old Standardbred gelding, born and raised in the Zanesville, Ohio, area, had been brought in from pasture in October, 1955, with patches of alopecia on the rump, back, and under the mane. Seven other horses, 2 to 4 years old, kept in the same field, showed no skin or hair abnormalities. During the three-month interval from October, 1955, to January, 1956, the lesions spread only slightly, but there was no tendency toward spontaneous healing.

On Jan. 13, 1956, the attending veterinarian* and one of the authors (C. J. F.) examined the infected gelding. Circular patches of alopecia, 2 to 3 inches in diameter, were found on the back, rump, shoulders, and under the mane. The central portions of the lesions were completely devoid of hair, and the skin was dry and scaly. There was no suppuration or kerion formation (fig. 1).

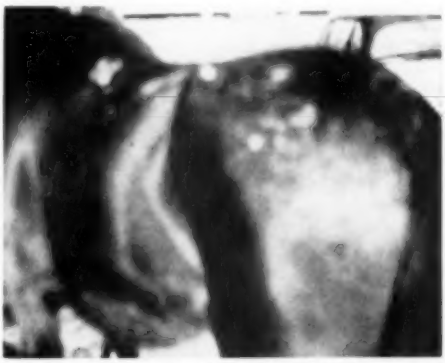

Fig. 1—Skin lesions produced by Microsporum gypseum.

Suspecting a fungous infection, hair and skin scrapings from the affected areas were collected and submitted to a laboratory† for confirmation. The laboratory diagnosis was ringworm infection. The animal was treated topically with three different proprietary fungicidal preparations. However, one month later, the horse died from a kidney infection. Therefore, it was not possible to evaluate the treatment. There was no transmission of the infection to the people who handled the horse nor to the other horses in the same field.

Mycological Findings.—Evidence of a dermatophyte infection was readily obtained at the laboratory by direct microscopic examination of the clinical material. This was accomplished by placing a few hairs and skin scrapings in a drop of 10

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†Mycology Laboratory of the Communicable Disease Center, Chamblee, Ga.

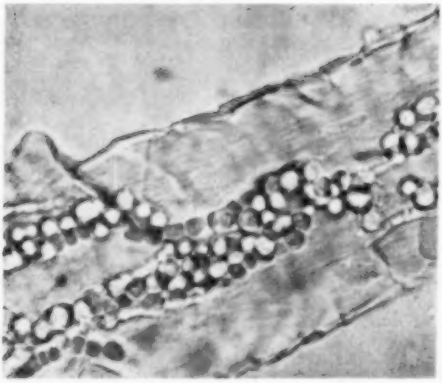

Fig. 2—Ectothrix pattern of hair invasion by Microsporum gypseum.

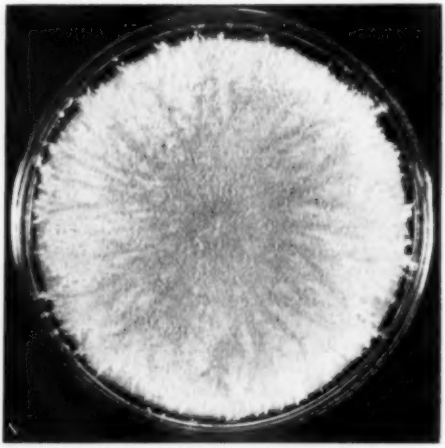

Fig. 3—Cultural appearance of Microsporum gypseum on Sabouraud's dextrose-agar containing cycloheximide, penicillin, and streptomycin.

per cent potassium hydroxide on a glass slide, adding a cover slip, and heating the slide gently over a Bunsen burner for a few seconds to hasten clearing. When examined under low and high power, a typical ectothrix pattern of hair invasion was observed. Large spores (5 to 8 μ) were found in chains and irregular masses on the surface of the hairs and mycelial filaments were seen inside the hairs (fig. 2). Examination of the clinical material under the Wood's lamp showed no fluorescence.

To isolate and identify the species of fungus causing the infection, the specimen was cultured on a special, selective-isolation medium containing the antibiotics cycloheximide, penicillin, and streptomycin.[12] After five days of incubation at 25 C., the dermatophyte that produced the infection was isolated in pure culture and identified on the basis of its gross and microscopic characteristics as *M. gypseum*. Grossly, the colony was powdery in texture and cinnamon brown in color, with a tan pigment on the reverse side (fig. 3). Direct microscopic

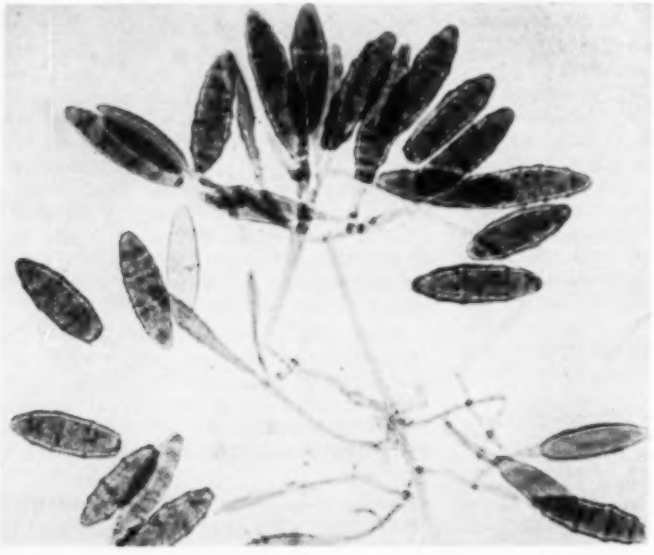

Fig. 4—Appearance of the diagnostic macroconidia of Microsporum gypseum culture.

examination of the mold showed many elliptic, multiseptate, rough-walled macroconidia (fig. 4). No microconidia were seen in this culture.

DISCUSSION

The apparent paucity of recorded cases of *M. gypseum* infection in horses should not be interpreted to mean that this infection is rare, nor that horses are highly resistant to this pathogen. A review of the world literature in 1953[1] showed that only 11 cases of *M. gypseum* infection in the dog had been reported; however, this infection is not as rare as was supposed.[13] During the three-month period ending Dec. 31, 1955, a total of 18 isolations of *M. gypseum* were made in this laboratory from dogs with clinical ringworm—almost twice as many as had been reported in the literature up to 1953. Possibly a similar situation exists among horses. Only by cultural studies of equine dermatophytoses can the extent and nature of *M. gypseum* infections in the horse be ascertained.

SUMMARY

The clinical and mycological findings in a horse spontaneously infected with *Microsporum gypseum*-induced ringworm are described. This is the first authenticated report of such an infection in this country.

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X-Ray Burns in a Horse.—Four exposures of x-rays (1,600 r), of 2 seconds each, to the hip region of a horse produced deep pigmentation, gangrene, and scab formation where the tube was in contact with the skin. The lesions were still present 27 months later.—*Vet. Bull., July, 1956.*

Localized Pulmonary Brucellosis.—Species of Brucella may produce chronic localized infections in a variety of tissues but rarely in the lungs. In three cases, two farmers and a farmer's wife, when radiographs of the chest revealed well-defined nodular lesions in the lungs and the latter were excised, *Brucella suis* was cultured from the lesions. Postoperatively, two of the three reacted serologically to *Br. suis*. All made satisfactory recoveries. In the 50-year-old woman, who had suddenly developed a nonprogressive hoarseness, the left vocal cord was fixed in complete adduction and the lesion was found adherent to the recurrent laryngeal nerves.—*J.Am.M.A., July 14, 1956.*

Another Test for Dwarfism.—Insulin injections for detecting bovine carriers of dwarfism have been tried at the University of Missouri. Leukocyte counts increase most and fastest in animals free of the dwarf gene, moderately in carriers, and little, if any, in dwarfs. The differences depend on the reaction of the adrenal gland to the insulin, thus indicating the activity of the pituitary gland.—*Hereford J., Aug. 15, 1956.*

Leptospira Survive in Soil.—Leptospira are reported, in Russia, to survive and multiply in mountain pastures where rainfall is heavy and the soil is retentive of moisture but is poor in carbonate and salts. The organisms do not survive the semi-desert summer conditions in the lowlands where the soil is rich in salt. In the latter, leptospirosis occurs only in the fall when the cattle are brought down from the mountains.—*Vet. Bull., July, 1956.*

Studies with Sulfonamide Combinations in Dairy Cattle

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WHEN USING systemic sulfonamide therapy, two important factors should be considered: (1) the establishment and maintenance of a blood level in excess of the minimum effective therapeutic level, and (2) minimizing the dangers of toxicity. Although sulfonamide toxicity may be manifested in several ways,[1] probably the most frequent one is renal damage caused by precipitation in the kidney tubules and pelvis.[2-6]

The first point is generally well accepted, as pointed out by E. K. Marshall[7] in his review article on the dosage schedule of chemotherapeutic agents. The matter of minimizing kidney toxicity was greatly facilitated, in 1945, by Lehr,[8] who showed the significantly greater solubility of sulfonamide mixtures, as compared with single sulfonamides. The greater the solubility of sulfonamide preparation, the less danger of renal damage.

Somewhat later, the question of the antibacterial effect of these mixtures was examined. In spite of the fact that *in vitro* tests indicated a decreased antibacterial activity of certain sulfonamide combinations,[9,10] these have nevertheless continued to find wide application in both human and veterinary medicine. Walker and Edmunds[11] reported on the clinical efficacy of a parenteral sulfamerazine-sulfamethazine mixture in the swine enteritis-pneumonia syndrome, and Walker[12] later discussed the use of the same preparation in various infectious diseases of cattle. As far as can be determined, there is no published information on either the solubility of the sulfonamide mixtures used by veterinarians or on the blood levels of these mixtures in cattle, with the exception of the work of Scheidy, McManus, *et al.*[13] on the sulfamerazine-sulfamethazine mixture. It is the purpose of this paper to consider the solubility, as well as the blood levels, of the following sulfonamide combinations in dairy cattle: A mixture of equal amounts of (1) sulfamerazine, sulfapyridine, and sulfamethazine (SMr-SP-SMT); (2) sulfamerazine-sulfapyridine and sulfathiazole (SMr-SP-ST); and (3) sulfamerazine-sulfamethazine (SMr-SMT).

EXPERIMENTAL METHODS

Solubility.—Sulfonamide solubility was determined by preparing saturated solutions of various individual sulfonamides, as well as the mixtures mentioned above. An excess of the drug to be tested was added to a series of McIlvaine's[14] buffers at pH 5.5 to 8.5. Following 12 to 24 hours of incubation at 37 C., the pH of the samples was checked with a Beckman Model G pH meter. They were then filtered and, after an appropriate dilution (1:10 to 1:50), the sulfonamide content was determined, using the Bratton-Marshall[15] method, with a Klett-Summerson photoelectric colorimeter. Sulfathiazole standards were employed throughout, and the results (table 1) represent solubility in milligrams per cubic centimeter of sulfathiazole. Under these conditions, the concentration of sulfonamide in the filtrate is equivalent to the solubility.

Blood Levels.—Healthy Holstein-Friesian cows, heifers, and calves were given 1 gr. per pound of body weight (143 mg./kg.) of the above mixtures intravenously. Oxalated blood samples were taken prior to injection and at regular intervals following administration. The sulfonamide content of these samples was determined by the aforementioned method. The preinjection samples were used as blood reagent blanks for standardizing the colorimeter. Sulfonamide standards of the mixtures being assayed were prepared in whole blood that was pooled from the animals used for each mixture. Use of such standards avoided the problem of recovery of sulfonamides from blood. The comparison of samples prepared from blood with water standards is not valid unless: (1) 100 per cent recovery of the drug is expected, or (2) a correction factor is used, as in the case of assaying sulfisoxazole[16] or sulfaquinoxaline. The results are shown (table 4 and graph 1). All samples were done in duplicate. For purposes of comparison, blood levels of sulfathiazole and sulfamethazine are also shown.

RESULTS AND DISCUSSION OF SOLUBILITY FACTORS

Table 1 shows the solubility of five commonly used sulfonamides and the three

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Paper No. 3530, scientific journal series, Minnesota Agricultural Experiment Station, St. Paul.

This work was supported in part by a grant from Sharp and Dohme, Inc., West Point, Pa.

TABLE 1—Sulfonamide Solubility in McIlvaine's Buffer (mg./100 ml. of Solution at 37 C.)

Initial pH	Final pH	SP	SMr	SMt	ST	SMr SMt	SMr SP ST	SMr SP SMt
5.5	5.5	46	34	56	107	88	162	123
6.5	6.5	52	55	60	118	106	184	141
7.5	7.3	60	143	99	234	221	341	255
8.5	8.0	84	264	228	374	424	576	474

SP = sulfapyridine, SMr = sulfamerazine, SMt = sulfamethazine, ST = sulfathiazole.

mixtures in aqueous buffers. These values are in agreement with those reported by Northey,[17] and they justify the following generalizations: (1) The sulfonamides are relatively insoluble compounds; (2) their solubility increases with increasing pH; and (3) the combination of two or more sulfonamides produces a solution with a total sulfonamide solubility that is significantly greater than any one sulfonamide alone. The total solubility approaches the sum of the solubilities of the individual components of a mixture. The implication of this is that sulfonamide mixtures would be safer from the standpoint of urinary tract precipitation. Studies on experimental animals (mice)[8] and clinical evaluation in man substantiate this.[18,19]

TABLE 2—Acetylation of Sulfonamides in Bovine Blood and Urine—Percentage of Total Amount in Blood and Urine

	Blood	Urine
Sulfanilamide	37.8	72.7[19]
Sulfathiazole	18.1	43.1[4]
Sulfapyridine	19.5	48.2[4]
Sulfadiazine	15.1	36.2[4]
Sulfamerazine	13.7	37.3[4]
Sulfamethazine	2.1-6.7[23]	20-30[23]
Sulfisoxazole	—	26[19]

*Reference cited

Sulfonamides undergo certain chemical changes in the body. These include oxidation, which may be followed by conjugation with glucuronic acid or sulfate, and acetylation. The recovery of oxidation products of sulfaquinoxaline from bovine urine indicates that sulfonamides may oxidize in the bovine body.[20] Further work is needed on this aspect of metabolism before its importance can be defined. However, several workers have shown that a significant degree of acetylation occurs in this species (table 2).

TABLE 3—Acetylsulfonamide Solubility (mg./100 ml. of Solution at 37 C.)

pH	SP[17]	ST[17]	SMr[23]	SMt[23]
5.5	11	10-20	33	70
6.5	19 (pH 7.1)	....	50	82
7.3-7.5	....	25-40	170	150
8.0-8.2	90	265	340	280

*Reference cited.

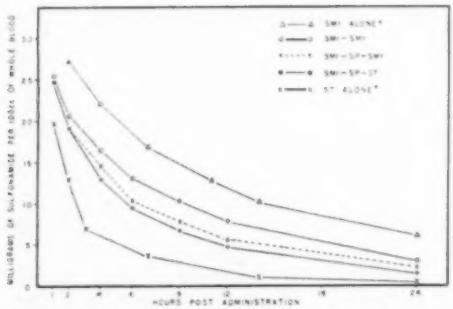
Since sulfonamides are acetylated to a significant degree in cattle, due consideration must be given to the solubility of this metabolite in the bovine urinary tract. Solubilities of the acetylated sulfonamides are given (table 3).

Acetylated sulfamerazine and sulfamethazine (sulfadiazine also) are more soluble than their parent compounds, while the converse is true for other sulfonamides. This indicates an advantage for sulfamerazine and sulfamethazine that the other sulfonamides do not have. Furthermore, the principle of increased total solubility of mixtures applies to the acetylated sulfonamides as well as to mixtures of the original or parent drugs.[24]

Sulfanilamide and sulfisoxazole are more soluble than the individual or mixed sulfonamides reported in the tables, but they have disadvantages that outweigh this factor. Sulfanilamide is more apt to give rise to other toxic reactions,[17] its antibacterial potency is below that for the other sulfonamides in common clinical usage,[25] and sulfisoxazole is incapable of maintaining effective blood levels for more than a few hours.[16]

TABLE 4—Blood Levels of Sulfonamide Mixtures (mg./100 ml. of Whole Blood)

Post-injection time	SMr - SP - SMt (0.33 gr./lb. of each) 12 animals		SMr - SP - ST (0.33 gr. /lb. of each) 12 animals		SMr - SMt (0.5 gr./lb. of each) 10 animals	
in hours	Average	Range	Average	Range	Average	Range
0	0.0	....	0.0	....	0.0	....
1	24.3	20.0 - 30.2	24.5	18.4 - 30.8	25.5	20.6 - 34.8
2	19.4	15.6 - 23.3	19.3	13.3 - 24.5	20.3	16.7 - 28.7
4	14.6	11.6 - 19.3	13.0	9.0 - 19.0	16.4	12.7 - 23.6
6	10.4	7.8 - 14.1	9.7	6.6 - 14.2	13.2	9.3 - 18.5
9	7.7	5.8 - 10.1	6.7	3.8 - 9.4	10.2	7.2 - 15.0
12	5.6	3.6 - 7.7	4.8	2.7 - 7.1	7.9	5.0 - 12.1
24	2.2	1.1 - 3.5	1.6	0.8 - 2.9	3.0	1.0 - 7.9



Graph 1—Blood levels of sulfonamide mixtures in cattle. SM†=sulfamethazine, SMr=sulfamerazine, SP=sulfapyridine, ST=sulfathiazole.

†Data taken from Jones, L. M., Smith, H. A., and Roepke, M. H., Am. J. Vet. Res., 10, (1949): 318.

RESULTS AND DISCUSSION OF BLOOD LEVELS

The blood levels obtained following intravenous injection of the three sulfonamide mixtures are recorded (table 4) and are reproduced graphically (graph 1). Mixtures of SMr-SP-ST and SMr-SP-SMT produce and maintain similar blood concentrations. These are in excess of 5 mg./100 ml. for about 12 hours. However, the sulfamethazine-sulfamerazine mixture at the same dose maintains levels of 5 mg./100 ml. or better for about 18 hours. This is in agreement with the work of others.[13]

To be effective, the sulfonamide blood concentrations should be maintained at therapeutic levels throughout the course of therapy. However, conclusive evidence as to what constitutes a therapeutically effective blood concentration is lacking. For man, most authorities consider concentrations of 8 to 15 mg./100 ml. as being necessary; for domestic animals, 5 mg./100 ml. is suggested[22] as the minimum effective therapeutic level. This value has come to be generally accepted by veterinary pharmacologists.

The problem of establishing and maintaining such concentrations in large animals is a difficult one. Sulfamethazine is the only sulfonamide available which will produce effective levels in cattle for 24 hours following a single dose of 1 gr. per pound. Other sulfonamides hold levels in cattle of 5 mg./100 ml. or better for approximately 18 to six hours in a descending order as follows: sulfamerazine, the sulfonamide mixtures, sulfapyridine, sulfanilamide, and sulfathiazole or sulfisoxazole.

Although the sulfonamide mixtures discussed in this paper possess a solubility advantage, they are inferior to sulfamethazine alone, with respect to blood levels. They are superior to sulfathiazole and sulfisoxazole, and are approximately equal to sulfanilamide, sulfamerazine, and sulfapyridine with regard to blood levels, depending on the particular mixture chosen. With the exception of sulfanilamide and sulfisoxazole, the mixtures are better with respect to solubility.

SUMMARY AND CONCLUSIONS

1) Sulfonamide mixtures are more soluble in urine and aqueous solution than are individual sulfonamides.

2) Mixtures of sulfathiazole-sulfapyridine-sulfamerazine and sulfamethazine-sulfapyridine-sulfamerazine maintain blood levels of 5 mg./100 cc. of blood or greater for about 12 hours following intravenous doses of 1 gr. per pound (143 mg./kg.).

3) A mixture of sulfamerazine and sulfamethazine maintains therapeutic blood levels for about 18 hours at intravenous doses of 1 gr. per pound (143 mg./kg.).

4) The relative importance of sulfonamide blood levels and solubility was briefly discussed.

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Trichinosis in Boys.—Nine of 14 boys, at a camp in North Carolina, who ate some commercial link sausage on April 22 became ill with trichinosis about 15 to 20 days later. Some of the sausage had not been well cooked, but none was available for examination later.—*Pub. Health Serv., June 14, 1956.*

Bone Marrow Counteracts Effects of Radiation.—The transfer of bone marrow from healthy rats to mice, at Oak Ridge National Laboratory, Tennessee, prevented death in a number of mice when done immediately after what otherwise would have been a fatal dose of radiation. The transfer of guinea pig bone marrow into mice also met with some success.—*Sci. News Letter, Aug. 18, 1956.*

Experimental Erysipelas in Poultry.—*In vitro* tests showed *Erysopelothrix rhusiopathiae* to be highly sensitive to penicillin, terramycin,® aureomycin,® and furazolidone; moderately sensitive to streptomycin and chloramphenicol; and completely resistant to a sulfonamide. *In vivo* tests, with chicks and turkeys, showed terramycin (100 mg./kg.) or benzathine penicillin (160,000 units/kg.) to be effective in therapy when given intramuscularly.—*Vet. Bull., Aug., 1956.*

Flies Transmit Swine Erysipelas.—Flies (*M. domestica*) allowed to feed on cultures of *Erysipelothrix rhusiopathiae*, were released with pigs whose skin had been scarified. Of the 14 pigs, 3 developed clinical erysipelas and 4 probably had a subclinical infection, since they were resistant later.—*Vet. Bull., Aug., 1956.*

Geese Carriers of Newcastle Disease.—That geese can be carriers of Newcastle disease was demonstrated when experimentally infected birds excreted the virus in their droppings, starting at three to six days; 15 days after the geese were infected, their droppings were still infective.—*Vet. Bull., July, 1956.*

Hypervitaminosis A in Pigs.—When an oily preparation containing 1,200,000 I. U. of vitamin A per gram was given *per os* to newborn pigs, many receiving 10,000,000 I. U. died and, on necropsy, showed marked changes in the kidneys.—*Vet. Bull., Aug., 1956.*

The fluorine eliminated in the urine equaled that ingested in the drinking water (at 1 p.p.m.) after the latter was consumed for one week by adults, but only after three to five years by children. This suggests that the maturity of the skeleton tissue influences its capacity to retain fluoride. There was no evidence of cumulative toxic fluorosis when drinking water was fluoridated at this level.—*Pub. Health Rep., Aug. 1956.*

Vesicular Exanthema in New Jersey.—On Aug. 14, 1956, V.E. was diagnosed in a herd of 1,300 hogs at Secaucus, N. J. All of the exposed hogs were slaughtered and strict quarantine measures imposed. This is the first appearance of V.E. since November, 1955, in California.—*U.S.D.A., Aug. 17, 1956.*

Nutrition, Zinc, and Parakeratosis

When healthy weanling pigs at the University of Wisconsin were fed corn, soybean meal, meat scraps, limestone, and salt (0.15% protein, 0.82% calcium, 0.47% phosphorus, 35 p.p.m. zinc), the addition of antibiotic or better quality protein had little effect on weight gains or on the incidence of parakeratosis.

When the calcium level was raised to 1.1 or 1.4 per cent, weight gains were decreased and the onset of parakeratosis increased. The addition of monosodium phosphate to the basic ration did not affect weight gains but decreased the skin lesions. When zinc was added, 50 p.p.m. greatly reduced but did not completely prevent parakeratosis, while 100 p.p.m. produced a marked increase in weight gain and essentially eliminated the skin lesions in nine weeks. The response to 500 p.p.m. of zinc was no greater than to 100 p.p.m.

When parakeratotic pigs were injected with inorganic zinc, the response was similar but the local reaction was severe.—*J. Anim. Sci., Aug., 1956.*

Zinc in the Tissues of Pigs

When zinc, as zinc sulfate, was added to the ration of pigs at the University of Wisconsin, it had no effect on the zinc content of the packed erythrocytes, spleen, intestine, or pancreas, but it did increase the zinc concentrations in the blood plasma, liver, and kidneys. When no zinc was supplemented, the addition of 2 per cent bone meal to the ration did not alter the zinc content of these tissues, but when 2 per cent bone meal plus 50 p.p.m. of zinc was added, the zinc content of the liver and kidneys was decreased, while that in the other tissues was unaffected.—*J. Anim. Sci., Aug., 1956.*

Sewage Sludge as Cattle Feed.— Dehydrated sewage sludge, when fed to steers, at Texas A. & M. College, as a substitute for cottonseed meal on an equal protein basis, resulted in slower gain and less efficient feed use.—*Feed Bag, Aug., 1956.*

Topical Fluorides and Tooth Decay.— Studies on the topical application of sodium fluoride and of stannous fluoride to the teeth of children indicated that the latter was more effective in preventing caries. When it was used continuously in a dentrifice (4,000 p.p.m.), tooth decay in children was reduced about 40 per cent over controls. Four topical applications at intervals by swab or spray also seemed beneficial but neither of these methods was as effective as water fluoridation throughout the period of tooth growth.—*Nutr. Rev., July, 1956.*

Diet and Urinary Calculi in Sheep

Eighty wether lambs (av. 76 lb.) showed no signs of urinary calculi after 100 days on grass-alfalfa hay. During the next 124 days, 7 of 20 lambs developed calculi when potassium was added to this ration; only 1 of 20 when phosphorus was added. When both were added, as potassium acid phosphate or as phosphoric acid and potassium carbonate, 28 of 40 lambs developed signs of calculi. When beet pulp alone was added to make 25 per cent of the ration, 26 of 40 developed signs of calculi, while beet molasses at 11 per cent had no effect. When phosphorus, potassium, and beet pulp were all added to the ration, 19 of 20 lambs developed calculi.—*J. Anim. Sci., Aug., 1956.*

Nutritional Anemia of Pigs

The blood of 209 pigs was studied until they were 84 days old. Mortality from anemia for the pigs kept on concrete was 12.7 per cent; on earth floors, 2.0 per cent. The cell volume range was of greater value in indicating dangerous anemia than the number of cells.—*Vet. Bull., April, 1956.*

Dried Rumen Contents Ineffective for Calves.—Using the artificial rumen technique, in Holland, dried rumen contents had no influence on the development of rumen flora and did not prevent calf scours, while fresh rumen transplants resulted in good cellulolytic flora within two weeks. The cellulolytic bacteria were killed during the drying process, but *Bacterium coli* and aerobic sporeforming organisms were not. —*Vet. Bull., Aug., 1956.*

Grass Tetany in Cattle.—Bovine grass tetany, in Holland, was least common in the spring when the temperature was above 58 F.; higher when the rate of plant growth was rapid; but highest when the temperature dropped below 58 F., especially after heavy rains at the end of summer.—*Vet. Bull., July, 1956.*

Veterinary Medical Writing

An eminent physician has said that the worst writing he sees is in medical publications. The responsibility for this, if true, must be shared by editors and authors.

Opinions vary as to what degree of literary excellence should be required before articles are accepted, and as to what degree editorial staffs should revise articles which are to be accepted.

When submitted, some manuscripts are excellent, others are like diamonds in the rough, and still others are highly polished but contain many flaws. Obviously, little can be done with the latter, but much can be done for the rough ones, although it requires painstaking effort. The question is, whose effort?

If inferior articles are published, whose reputation suffers most—the author's, the editor's, or the profession's? Also what would be the effect on future writing if inaccuracies in anatomical descriptions and technical terms, or sloppy organization, poor grammar, and jargon were accepted?

In this day of prolific writing, editors are responsible for seeing that a maximum of useful information is presented in a minimum of space and in an understandable form. An author should be allowed to use his preferred style of expression, so long as it is grammatically and technically correct, but should he expect to be allowed superfluous words, phrases and repetition?

Authors vary from the eager to the reluctant. Some doubtless feel under pressure to produce articles. Unfortunately, experienced practitioners are too often in the reluctant group; the same men who are generous with direct aid, when requested by their colleagues, usually hesitate to express themselves in writing.

To paraphrase an editorial in the *Journal of the American Medical Association* (vol. 155, p. 1573), the physician [or veterinarian] should know how to prepare a paper since he has a moral obligation to disseminate his observations for the benefit of mankind. Furthermore, preparing a paper broadens one's knowledge, since it requires research and, thus, better fits the author to serve his patients. To quote Sir Francis Bacon—"Reading maketh a full man, conference a ready man, and writing an exact man." The reviewing of literature should also suggest how to write reports.

Practitioners who wish to submit independent articles will find our editorial staff willing to aid in their preparation. Just be sure all pertinent data are submitted. The article will be returned to the contributor for approval before being published.

Preparation of an Article

Much has been written on the preparation of medical articles. Some of the suggestions follow.

First, define the subject so that irrelevant material need not be considered. Then review the available literature and compare it with your data, so that duplication can be avoided. If it still seems advisable to write the article, make an outline of what should be included.

The *title* should be concise but must indicate the contents.

The *opening paragraph* should attract and prepare readers for what is to follow.

The *body* of the article should systematically present all pertinent information but should avoid the irrelevant and unimportant. For instance, a report of a new method of intestinal anastomosis should clearly describe any unique preparatory measures, all details of surgery on the intestine, and any special features of the aftercare. It should not be burdened with routine procedures; the readers probably have or can get that information.

Illustrations add value and often make lengthy descriptions unnecessary. Information often can be condensed into simple tables, but long tables are boring and often confusing; if the authors can't summarize the latter, most readers won't.

Most articles require a *discussion* and also *conclusions* or a *summary*, or both. Conclusions, when presented, should briefly enumerate the author's evaluation of the procedures reported. The summary should crisply list only the important findings presented—it should not describe the article. While research articles usually require a more elaborate outline than the above, case reports and short articles may need none.

Preparation of a good article always requires several revisions, preferably with an interval of several days between efforts. We should heed Voltaire who, in apologizing for a long letter, said, "If I had more time, I would write you a short one."

Current Literature

ABSTRACTS

Studies in Deaths of Young Ruminants

Listeria monocytogenes added to the drinking water of 2 pregnant ewes and 3 pregnant goats produced fatal listeric infection in the young manifested by abortion, if exposure was early in gestation, or by death of the young within the first postnatal week, if exposure was near term. Two dams died from listeric septicemia following exposure. One pregnant goat was infected by contact with the exposed goats and aborted fetuses.

Lesions in the fetuses and in the young born at term appeared to be identical to those seen in natural outbreaks of listeric abortion or perinatal death. Of 57 isolations of *L. monocytogenes* from fetuses and membranes, seven were positive only after refrigeration of the tissue suspensions. A goat exposed by conjunctival instillation late in gestation died of listeric encephalitis. Three normal young were delivered by cesarean section. Listeric abortion may be a public health hazard.—[*M. L. Gray, Chintamani Singh, and Frank Thorp, Jr.: Abortion and Pre- or Postnatal Death of Young Due to Listeria Monocytogenes. III. Studies in Ruminants. Am. J. Vet. Res., 17, (July, 1956): 510-516.*]

Trichloroethylene-Extracted Feeds

I. Acute Aplastic Anemia in Young Heifers

The biological effects of feeding trichloroethylene-extracted soybean oil meal (TCESOM) to various species is discussed. The TCESOM was an industrial product made by extracting the fat from crushed soybeans with trichloroethylene. The unextracted residue thus prepared from one specimen of soybeans, when fed to young heifers at a level of 3 lb. per day, induced acute aplastic anemia which caused death in 35 to 55 days. The syndrome was indistinguishable from that observed on farms where this type of feed had been used.

After approximately 25 days of feeding, the heifers developed thrombocytopenia, leukopenia, neutropenia, a relative lymphocytosis, anemia, and a severe hemorrhagic syndrome. There were marked differences in the degree of toxicity produced by two different specimens of TCESOM.—[*W. R. Pritchard, C. E. Rehfeld, N. S. Mizuno, J. H. Sautter, and M. O. Schultze: Studies on Trichloroethylene-Extracted Feeds. I. Experimental Production of Acute Aplastic Anemia in Young Heifers. Am. J. Vet. Res., 17, (July, 1956): 425-429.*]

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II. Chronic Aplastic Anemia in Heifers

Different specimens of trichloroethylene-extracted soybean oil meal (TCESOM) were fed to young heifers continuously or intermittently. Without exception, they induced a temporary or permanent leukopenia and thrombocytopenia. Death from aplastic anemia occurred as late as 563 days after the start of continuous daily feeding of 1 lb. of a toxic specimen of TCESOM.

Aplastic anemia of varying degrees of severity can be induced by feeding different levels of TCESOM. Soybeans processed with trichloroethylene within a few months after harvest yielded a meal of a high degree of toxicity. Processing of old soybeans yielded a meal with a low but detectable degree of toxicity.—[*W. R. Pritchard, C. E. Rehfeld, W. E. Mattson, J. H. Sautter, and M. O. Schultze: Studies on Trichloroethylene-Extracted Feeds. II. The Effect of Feeding Different Levels of Trichloroethylene-Extracted Soybean Oil Meal to Young Heifers—Experimental Production of Chronic Aplastic Anemia. Am. J. Vet. Res., 17, (July, 1956): 430-436.*]

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III. Toxicity in Young Calves

The young calf is a suitable experimental animal for the study of various aspects of intoxication by trichloroethylene-extracted soybean oil meal (TCESOM). Soybean products *per se* were not toxic to calves but specimens of TCESOM, known to be toxic to older cattle, caused death of calves from acute aplastic anemia as early as 24 days after the start of feeding. The signs and lesions of intoxication were essentially the same in calves as in older cattle. The principal difference was that the erythrocyte count and the hemoglobin concentration of the blood remained essentially normal in the calves.

Trichloroethylene, at levels far exceeding those present as residual trichloroethylene in TCESOM, was nontoxic when fed to calves for as long as 346 days. The toxic factor present in some specimens of TCESOM could not be detected in soybean oil or in the phosphatide fraction. By administering the total ration to a calf through an abomasal fistula, it was shown that digestion of the toxic feed in the rumen is not necessary to produce fatal aplastic anemia. No allergic reaction of calves to the toxic feed could be demonstrated.—[*W. R. Pritchard, W. E. Mattson, J. H. Sautter, and M. O. Schultze: Studies on Trichloroethylene-Extracted Feeds. III. The Use of Young Calves for Study of Various Aspects of Toxicity of Trichloroethylene-Extracted Soybean Oil Meal. Am. J. Vet. Res., 17, (July, 1956): 437-440.*]

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IV. Toxicity in the Horse

Two horses were fed a specimen of trichloroethylene-extracted soybean oil meal (TCESOM) which was known to be highly toxic to cattle. Both animals developed aplastic anemia and died after consuming 1,000 to 1,100 lb. of TCESOM over periods of 198 and 287 days, respectively. The clinical signs, blood changes, and pathological changes were indistinguishable from those that occur in TCESOM intoxication in cattle. Susceptibility to TCESOM intoxication is not restricted to ruminants. The feeding of as much as 6 lb. of hexane-extracted soybean oil meal per day to a

horse during eight months produced no harmful effects.—[*W. R. Pritchard, Robert Hammer, J. H. Sautter, and M. O. Schultze: Studies on Trichloroethylene-Extracted Feeds. IV. Susceptibility of the Horse to the Toxic Factor in Trichloroethylene-Extracted Soybean Oil Meal. Am. J. Vet. Res., 17, (July, 1956): 441-443.*]

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V. No Toxic Factor in Milk of Cows Fed TCESOM

Three heavily lactating cows were fed a ration containing a relatively large amount of trichloroethylene-extracted soybean oil meal (TCESOM) for 62 to 106 days. All developed blood changes and clinical signs that are characteristic of aplastic anemia of cattle. One cow died on the 118th day, 24 days after feeding of TCESOM had been stopped. After the tenth day, *i.e.* when cows were under the influence of the toxic factor, their milk was fed to 4 dogs, 4 cats, and 4 calves for 90 to 96 days as the principal component of their diet. None of these animals showed clinical or hematological abnormalities and no lesions were found at necropsy.—[*W. R. Pritchard, W. E. Mattson, J. H. Sautter, and M. O. Schultze: Studies on Trichloroethylene-Extracted Feeds. V. Failure to Demonstrate the Presence of a Toxic Factor in the Milk of Cows Fed Toxic Specimens of Trichloroethylene-Extracted Soybean Oil Meal. Am. J. Vet. Res., 17, (July, 1956): 444-445.*]

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VI. Effects of Feeding TCESOM to Sheep

A specimen of trichloroethylene-extracted soybean oil meal (TCESOM) which was known to be highly toxic to cattle was fed to 2 lambs, 4 open ewes, and 4 pregnant ewes in amounts up to 1.8 lb. per day. This feed produced illness and signs of blood dyscrasia. The lambs died after 38 and 56 weeks and 5 ewes died after ten, 18, 32, and 56 weeks, respectively. Three ewes survived 351 days on the TCESOM ration and 26 subsequent weeks on a ration which did not contain TCESOM.

Four control animals fed hexane-extracted soybean oil meal remained normal for 78 weeks. The hematological changes observed in the sheep fed TCESOM were not always consistent with those associated with aplastic anemia. If the same factor present in TCESOM is responsible for the symptoms observed in cattle and in sheep, it appears that sheep are much more resistant to its toxic effects.—[*W. R. Pritchard, V. Perman, W. E. Mattson, J. H. Sautter, and M. O. Schultze: Studies on Trichloroethylene-Extracted Feeds. VI. The Effects of Feeding Trichloroethylene-Extracted Soybean Oil Meal to Sheep. Am. J. Vet. Res., 17, (July, 1956): 446-447.*]

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VII. Observations of Laboratory Animals Fed TCESOM

A specimen of trichloroethylene-extracted soybean oil meal which is highly toxic to cattle was fed for long periods at high levels to mice, rats, hamsters, guinea pigs, rabbits, and dogs. In contrast to horses and cattle, none of these species showed any evidence of aplastic anemia or other marked blood dyscrasia. Feeding of 72.9 per cent of the TCESOM ration to guinea pigs invariably produced a syndrome of gradual, generalized debilitation which terminated in death. Guinea pigs fed the same proportion of hexane-extracted soybean oil meal gained weight and remained normal. Feeding of 72.9 per cent of the TCESOM ration to dogs induced the gradual development of anorexia, weight loss, a generalized debilitation, and ultimate death (after 378 to 438 days). Feeding of the same proportion of hexane-extracted soybean oil meal to dogs for 500 days had no detectable harmful effects. No marked toxic effects of TCESOM could be demonstrated with mice, rats, hamsters, or rabbits. It is not known whether the factor in TCESOM which causes debilitation and death in guinea pigs and dogs is the same as that which causes aplastic anemia in cattle and horses.—[*W. R. Pritchard, F. Sauer, C. E. Rehfeld, V. Perman, J. H. Sautter, Shohachi Wada, and M. O. Schultze: Studies on Trichloroethylene-Extracted Feeds. VII. Observations with Laboratory Animals Fed Trichloroethylene-Extracted Soybean Oil Meal. Am. J. Vet. Res., 17, (July, 1956): 448-454.*]

Macroscopic Anatomy of the Canine Ear

By the use of modern techniques, the gross morphology of the ear of the dog has been reinvestigated. Particular attention was given to the names of the parts of the external ear and the inter-relationship of structures of the internal and middle ears. All of the eight detailed drawings were made from representative dissections or special preparations.—[*R. Getty, H. L. Foust, E. T. Presley, and M. E. Miller: Macroscopic Anatomy of the Ear of the Dog. Am. J. Vet. Res., 17, (July, 1956): 364-375.*]

Dog and Cat Behavior

Breed selections for the capacity to perform specialized tasks have been carried further in the dog than in any other species. Cats, too, have a considerable variability in behavior. At Jackson Memorial Laboratory, more than 3,500 dogs and 600 cats have been produced and studied for the past ten years. Information of interest to all handlers of cats and dogs is presented.—[*Edna M. Du Bois: Dog and Cat Behavior. Proc. Animal Care Panel (1955): 47-51.*]—N. R. BREWER.

Serological Studies of Canine Distemper

This paper is a continuation of a study on jaundice and bilirubinemia in canine distemper in raccoons and ferrets. Spleens from infected animals, taken at the height of illness, contained distemper virus antigens demonstrable in complement-fixation tests. Although a rise of circulating complement-fixation antibodies could be shown in serums of ferrets between the time of inocula-

tion and time of death from acute illness, this situation did not develop in raccoons. Only raccoons that survived illness had demonstrable circulating antibodies. Neutralization tests performed by intracerebral inoculation of suckling mice proved useful in confirming results.—[*L. Kilham: Serological Studies of Canine Distemper—Complement-Fixation with Spleen Antigens. Am. J. Vet. Res., 17, (July, 1956): 398-401.*]

Effect of Ammonium Sulfate on Salmonella Gallinarum

Suspensions of 41 strains of *Salmonella gallinarum* and 23 strains of standard type *Salmonella pullorum* were exposed to varying concentrations of ammonium sulfate, incubated for a short time, and then centrifuged. It was found that an ammonium sulfate concentration of 265 Gm. per liter completely cleared the supernatant fluids of most *S. gallinarum* suspensions, but had considerably less effect on the turbidity of suspensions of standard type cultures of *S. pullorum*. These observations suggest that there may exist minor antigenic differences between *S. gallinarum* and standard strains of *S. pullorum* that are not demonstrable by conventional serological methods.—[*J. E. Williams and M. E. Harris: Antigenic Studies Using Ammonium Sulfate. IV. The Sedimentation Effect of Ammonium Sulfate on Salmonella Gallinarum. Am. J. Vet. Res., 17, (July, 1956): 535-537.*]

Antigenic Studies of the Ornithosis Virus

The intradermal allergy test was developed to be used as a tool in determining the extent of ornithosis infection in turkeys and chickens. A soluble skin test antigen extracted from the ornithosis virus was more active for the detection of allergy in ornithotic chickens than other antigens tested.

Positive intradermal (wattle) reactions were given by 131 of 142 experimentally infected chickens. Nonspecific reactions in normal chickens were rare. Repeated intradermal inoculations in normal chickens stimulated temporary production of low titer circulating antibody, but did not lead to a state of cutaneous hypersensitivity.—[*Albert A. Benedict, Edith O'Brien, and Clarence McFarland: Antigenic Studies on the Psittacosis-Lymphogranuloma Venereum Group of Viruses. III. Detection of Ornithosis Hypersensitivity in Experimentally Infected Chickens. Am. J. Vet. Res., 17, (July, 1956): 543-546.*]

Estrogen Contamination in Animal Feed

Estrogen contamination in feed interfered with the mouse uterine response for the detection and quantitative assay of estrogens by a method that had given good results for 15 years prior to contamination.—[*R. I. Dorfman, and A. S. Dorfman: Estrogen Contamination in Animal Feed. Proc. Animal Care Panel (1955): 16-18.*]—N. R. BREWER.

Effect of Antibiotics and Chemical Agents on PPLO

A method is described for the evaluation of drugs in the embryonating chicken egg. The infectious agent was inoculated into the yolk sac and the drug inoculated into the allantoic cavity. The activity of several chemical agents tested against an antibiotic-sensitive strain of PPLO by the *in ovo* method was barely significant. Erythromycin and viridogrisein were the most active, with the tetracycline drugs comparable in activity but less active, and streptomycin and dihydrostreptomycin the least active.

In vitro antibiotic studies with several strains of PPLO of avian origin showed tetracycline and oxytetracycline as the most active antibiotics, with erythromycin, chlortetracycline, and streptomycin comparable but not as active. Dihydrostreptomycin was the least active of the six antibiotics tested. Differences to antibiotic sensitivity were observed with PPLO of avian origin by both *in vitro* and *in ovo* methods of evaluation.—[*R. Yamamoto and H. E. Adler: The Effect of Certain Antibiotics and Chemical Agents on PPLO of Avian Origin. Am. J. Vet. Res., 17, (July, 1956): 538-542.*]

FOREIGN ABSTRACTS

Treatment of Bovine Coccidiosis with Ammonium Sulfate

On the theory that bases in the large intestine would react with ammonium salts to librate ammonia, which is toxic to oöcysts, the authors treated coccidiosis in calves and yearlings with ammonium chloride and ammonia sulfate. Five of the calves were artificially infected with *Eimeria zurni*. The species involved in the natural disease are not named. The sulfate was preferred because it was less irritant and more stable than the chloride. The dose was 3 to 5 Gm. of ammonium sulfate with 2 ml. of lactic acid given in the milk twice daily. The hemorrhagic diarrhea stopped in one to four days and all of the 175 animals treated recovered. A controlled trial in an infected herd indicated that the drug was also effective as a prophylactic measure.—[*V. A. Petrov, Rostov Vet. Res. Sta.: The Treatment and Prophylaxis of Bovine Coccidiosis with Ammonium Salts. Veterinariya, 33, (Feb., 1956): 30-32.*]—ROBERT E. HABEL.

Complement-Fixation Test for Vibriosis

A modification called the long complement-fixation test was found to be more sensitive than the agglutination tests or the standard complement fixation. The main differences from the standard method were: (1) The serum to be tested was inactivated at 63 C. for 30 minutes. (2) The complement was not titrated but was used in a constant dilution of 1:30, and in 0.5-ml. quantities. (3) The hemolytic system was titrated against negative serum to determine the maximum amount that was completely hemolyzed. This is the measure of the

activity of the complement. (4) Fixation of complement was carried out at 0 to 4 C. for 16 to 18 hours.—[*P. A. Trilenko, Leningrad Vet. Res. Inst.: A New Serologic Reaction for the Diagnosis of Vibriosis in Cattle—LCFT. Veterinariya, 33, (Feb., 1956): 70-77.*]—ROBERT E. HABEL.

BOOKS AND REPORTS

Course in Veterinary Microbiology

Shortly after the fourth edition, this manual was revised and the fifth edition published.

As an introduction, the regulations and precautions in the microbiological laboratory are discussed. These are particularly important for students dealing with infectious material.

Further chapters are devoted to the microscopical technique and to the morphological and cultural examination of the infectious material. Special attention is given to the animal experiments and serological methods used for the diagnosis of various infectious disorders.

The main part of the manual deals with the survey of the Hyphomycetes and Schizomycetes, including Blastomyces, molds, fungi imperfecti, cocci, and bacteria. The important protozoa and viruses, are also discussed.

In the final chapter, there are prescriptions for the fundamental staining solutions and culture mediums.

The manual is supplemented with instructive colored illustrations and good tables. It has been written clearly and distinctly and is a valuable help not only for the students but also for veterinary practitioners.—[*Course in Veterinary Microbiology. (Kursus der Veterinärmedizinischen Mikrobiologie). By Kurt Wagener. 5th ed. 161 pages. 44 illustrations and 18 tables. Paul Parey, Berlin and Hamburg, Germany. Price D.M. 26.80.*]—F. KRAL.

Medical Parasitology

The second edition of this brief text is about 50 pages longer than the one published in 1950. It discusses protozoa, nematodes, tapeworms, flukes, and arthropods. This is followed by a discussion of treatment of laboratory techniques, a synopsis, and a glossary. Except for a color plate of malaria parasites, all the illustrations are drawings, many more or less diagrammatic.

The author is to be commended for the direct, easily grasped style in which the book is written.

This is not a complete reference work, but a medical school text. In general, only the more common parasites are discussed. Clinical and laboratory diagnostic methods and treatment are emphasized. Despite a number of minor errors, the book is adequate for its purpose.

This book is intended for medical students and practicing physicians and is of little value to veterinarians or veterinary parasitologists. Except for well-known parasites like Trichinella, Echinococcus, and Taenia, very little is said about the public health aspects of parasitism in domestic animals. There is no mention, for example, that *Strongyloides stercoralis* occurs in dogs and cats in addition to man, nor is the recent work on visceral larva migrans of young children due to Toxocara larvae referred to.—[*Medical Parasitology for Medical Students and Practicing Physicians. By William G. Sawitz. 342 pages. The Blakiston Division, McGraw-Hill Book Co., Inc., New York, 1956. Price $6.00.*]—NORMAN D. LEVINE.

Antibiotics in Agriculture

This book is a compilation of the papers and panel discussions presented at the First International Conference on Antibiotics in Agriculture held Oct. 19-21, 1955, in Washington, D.C. The subjects, which include growth response in animals, special biological problems, mode of action, crop usage, food preservation, and public health aspects, represent a survey of what has been learned, in this and other countries, of the effects of antibiotics on animal and plant growth and on food preservation, while pointing up the need for further research on this important subject.—[*Proceedings of the First International Conference on the Use of Antibiotics in Agriculture. National Academy of Sciences—National Research Council, Washington 25, D. C. 1956. Price $2.50.*]

Handbook of Poisons

This pocket-sized publication presents a concise summary of various products poisonous to human beings. The poisons are classified as pesticides and other agricultural poisons, industrial hazards, household chemicals, medicinal poisons, and plant and animal hazards. Where possible, the author has grouped pharmacologically related agents. For each poison described, the diagnosis, emergency treatment, prophylaxis, and prognosis are given.

Sections of the book have application to the veterinary field, particularly the agricultural poisons (insecticides, pesticides, rodenticides), plant and animal hazards (reptiles, arachnids and insects, fish, plants), and medicinal agents (analgesics and antipyretics, anesthetics, depressants, etc.)—[*Handbook of Poisons. By Robert H. Dreisbach. 426 pages, including 20-page index. Illustrated. Lange Medical Publications, Los Altos, Calif. 1955. Price $3.00.*]

Insects and Spiders

This simplified, yet scientific, classification of insects and spiders was written to facilitate teaching first-year biology classes. Insects are classified according to families, spiders according to families and genera, and pertinent information is given in each instance. In general, the species described are those found in southern England.

The material is well organized and is supplemented with numerous drawings to help differentiate and identify specimens.—[*Insects and Spiders. By C. P. Friedlander and D. A. Priest. 124 pages. 21 plates. Philosophical Library, Inc., New York, N. Y. 1956. Price $2.75.*]

THE NEWS

Dr. Hurst Completes Research Fellowship

Dr. Harold L. Hurst (TEX '48), recipient of an AVMA Research Fellowship in November, 1955, was awarded the Ph.D. degree by Purdue University in June, 1956. The subject of his thesis was "Studies of Some Enteritides

Dr. Harold L. Hurst

of Swine." Three articles, under the title of "Studies on Therapy of Swine Enteritis," by Drs. Hurst and Doyle appeared in the July 1 issue of the JOURNAL. They were taken from the thesis submitted by Dr. Hurst in earning his previous degree, an M.S., at Purdue University in August, 1954.

Dr. Hurst, who is now an associate veterinary pathologist on the staff of the Department of Veterinary Science of the Louisiana State University, Baton Rouge, was born at Dallas, Texas, Feb. 25, 1919. His veterinary training, begun at the A. & M. College of Texas in 1940, was interrupted by three years of service in the Medical Department, Veterinary Service, of the Army Air Force. He completed the course for his D.V.M. degree in February, 1948.

After a year in practice, Dr. Hurst accepted a position with Pitman-Moore Company at Zionsville, Ind. Four years later, he resigned to begin his graduate studies at Purdue University.

Dr. Hurst now resides, with his wife and two daughters, at 685 Hart Drive, Baton Rouge, La.

Veterinarian Trainee Examination Announced by U. S. Civil Service

An examination open to junior veterinary students has been announced by the U. S. Civil Service Commission. Trainees will be employed during summer vacation in Agricultural Research Service, U.S.D.A., at rate of $3,670 a year during training period; after graduation from veterinary college, they will be eligible for positions at $5,200 a year.

Applications will be accepted until Nov. 6, 1956, by the Board of Civil Service Examiners, U. S. Department of Agriculture, Washington 25, D. C.

U. S. GOVERNMENT

Veterinary Personnel Changes.—The following changes in the force of veterinarians in the U.S.D.A. are reported as of Sept. 4, 1956.

TRANSFERS

James R. Coler, from Cincinnati, Ohio, to Columbus, Ind.

James W. Connelly, from New York, N. Y., to Los Angeles, Calif.

James A. Faughn, from Springfield, Ill., to St. Louis, Mo.

Roscoe G. Feller, from Bentonville, Ark., to DeQueen, Ark.

LeRoy T. Fisher, from Baton Rouge, La., to Little Rock, Ark.

Charles W. Freudenberg, from Columbus, Ind., to Fort Branch, Ind.

COMMENCEMENTS

University of Pennsylvania.—At the 1956 commencement exercises of the School of Veterinary Medicine, University of Pennsylvania, Philadelphia, the following 45 candidates were presented for the D.V.M. degree:

Elizabeth B. Atwood
Bruce S. Blauch
Gerald Borman
Daniel M. Burnside, III
William F. Butler
John W. Cable
Richard O. Cook
Wendell L. Cooper
Helena J. Costantini
Daniel Fasnacht
Lorraine Fournier
George A. Franko
Ellen A. Friedman
Karl L. Gabriel
Charles H. Garvin
Edwin Gearhart
William Jackson
Charles Knecht
Paul Kozak
Denzil Lee
Peter A. Lien
Anthony Lisehora
Joseph G. Lopatofsky
Raymond Lundberg
Edward T. Mallinson
Alan Morse
John Munnell
Warren Piersol
Wesley G. Pietz
Harry A. Reynolds
Richard R. Ryan
Jay Simmons
Thomas V. Sollas, Jr.
Cecil Sprung
Paul J. Suorsa
Robert J. Tashjian
John R. Terry
Richard A. Vaclavik
Max Van Buskirk, Jr.
George Vernimb
Donald Wampler
Stanley N. Wampler
Arthur B. Whitney
Edward K. Wojciechowski
Ted Yuhas

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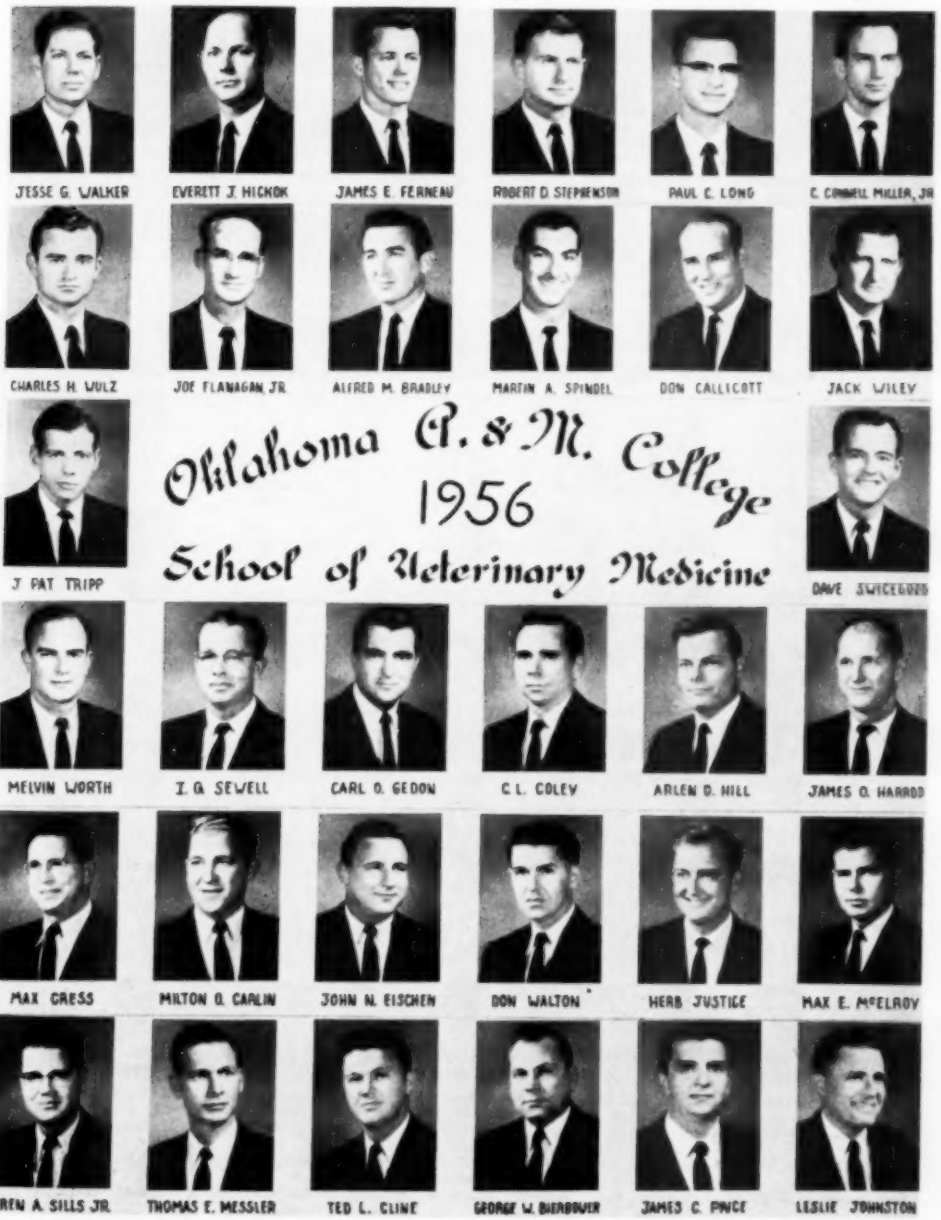
Oklahoma A. & M. College.—At the 1956 commencement exercises of the School of Veterinary Medicine, Oklahoma A. & M. College, Stillwater, the following 32 candidates were presented for the D.V.M. degree:

George W. Bierbower
Alfred M. Bradley
Don Callicott
Milton O. Carlin
Ted L. Cline
C. L. Coley
Max Cress
John N. Eischen
James E. Ferneau
Joe Flanagan, Jr.
Carl O. Gedon
James O. Harrod
Everett J. Hickok
Arlen D. Hill
Leslie Johnston
Herb Justice
Paul C. Long
Max E. McElroy
Thomas E. Messler
C. Connell Miller, Jr.
James C. Price
I. G. Sewell
Oren A. Sills, Jr.
Martin A. Spindel
Robert D. Stephenson
Dave Swicegood
J. Pat Tripp
Jesse G. Walker
Don Walton
Jack Wiley
Melvin Worth
Charles H. Wulz

Graduating Class, 1956, School of Veterinary Medicine, Oklahoma A. & M. College



Texas A. & M. College.—At the 1956 commencement exercises of the School of Veterinary Medicine, Texas A. & M. College, College Station, the following 54 candidates were presented for the D.V.M. degree:

J. W. Ard, Jr.
J. R. Arledge
O. B. Beard, Jr.
D. G. Black
F. N. Black
R. H. Brock
J. P. Broussard
W. H. Buller
W. E. Bush
R. R. Caldwell
J. K. Callan
B. W. Cline
J. D. Coleman
J. R. Collins
J. K. Cooper
L. F. Cox
H. C. Dismukes, Jr.
R. T. DuBose
A. D. Dubon
T. T. Elrod
G. C. Green
A. L. Griffith
J. E. Gross
D. H. Guiles
E. O. Harrison
T. G. Hildebrand
E. M. Himes, Jr.
J. W. Holcombe
A. F. Hopkins, Jr.
J. W. Kyle
J. P. Lancaster
W. R. Larson
Winfield Massie
Edward Michelitch
T. O. Miessler
M. G. Munster
W. F. Norvell
J. R. Pavelka
B. J. Payne
J. R. Pearce
T. A. Richard
J. P. Robisheaux
W. E. Roe, Jr.
J. M. Rucker
C. J. Shepler, Jr.
E. T. Skidmore
R. C. Thomas, Jr.
G. G. Tolbert
B. R. Trimmier
J. R. Watkins
J. E. West
D. W. Wisdom
R. L. Younger
M. P. Zimicki, Jr.

AMONG THE STATES AND PROVINCES

Florida

West Coast Association.—At a recent meeting of the West Coast Veterinary Medical Association, the following officers, all of St. Petersburg, were elected for the ensuing term: C. C. Murphy, president; R. F. Minnick, presi-

Graduating Class, 1956, School of Veterinary Medicine, Texas A. & M. College

Top row (left to right)—J. W. Ard, Jr., J. R. Arledge, O. B. Beard, D. G. Black, F. N. Black, R. H. Brock, J. R. Broussard, W. H. Buller, W. E. Bush.

Second row—R. R. Caldwell, J. K. Callan, B. W. Cline, J. D. Coleman, J. R. Collins, J. K. Cooper, L. F. Cox, H. C. Dismukes, Jr., R. T. DuBose.

Third row—A. D. Dubon, T. T. Elrod, G. C. Green, A. L. Griffith, J. E. Gross, D. H. Guiles, E. O. Harrison, T. G. Hildebrand, E. M. Himes, Jr.

Fourth row—J. W. Holcombe, A. F. Hopkins, Jr., J. W. Kyle, J. P. Lancaster, W. R. Larson, Winfield Massie, Edward Michelitch, T. O. Miesslar, M. G. Munster.

Fifth row—W. F. Norvell, J. R. Pavelka, B. J. Payne, J. R. Pearce, T. A. Richard, J. P. Robisheaux, W. E. Roe, Jr., J. M. Rucker, C. J. Shepler, Jr.

Sixth row—E. T. Skidmore, R. C. Thomas, Jr., G. G. Tolbert, B. R. Trimmier, J. R. Watkins, J. E. West, D. W. Wisdom, R. L. Younger, M. P. Zimicki, Jr.

dent-elect; and W. F. Casler, secretary-treasurer.

s/W. F. CASLER, *Secretary.*

Maryland

State Association.—At a meeting of the Maryland State Veterinary Medical Association, held on June 28-29, 1956, at the Hotel George Washington, Ocean City, the following speakers and their subects were included in the program: Charles W. Raker, University of Pennsylvania School of Veterinary Medicine, Philadelphia (anesthesia of the bovine foot, lameness in the horse); Jean Holzworth, Boston, Mass. (diseases of the cat); John McGrath, University of Pennsylvania School of Veterinary Medicine, Philadelphia (nervous disorders, intracranial pathology of the dog); W. M. Moulton, ARS, U.S.D.A., Baltimore (muzzle disease); and A. H. Craige, Jr., Pitman-Moore Co., Indianapolis, Ind. (biological therapy in mastitis).

A panel discussion of gastrointestinal disorders in cattle was presented by David Rice, Warren, Pa.; Dale Wiley, Jarrettsville, Md.; Harold Schaden, Frederick, Md.; Richard Sears, Cazenovia, N. Y.; and Marvin Jacobs, Chestertown. Drs. Silas Richman, Irwin Neserke, and Douglas Burley, all of Baltimore, discussed small animal practice.

At the business session, the following officers were elected for the 1956-1957 term: Harold Schaden, Frederick, president; Thomas Ladson, Jr., Olney, first vice-president; Dale

s/JOHN D. GADD, *Secretary.*

New Mexico

State Association.—The New Mexico Veterinary Medical Association held its annual convention in Portales, on Sept. 11-12, 1956.

The following were program participants: A. H. Craige, Jr., Pitman-Moore Co., Indianapolis, Ind. (sex hormones); Charles E. Deyhle, Clarendon, Texas (small town practice); O. R. Adams, Colorado A. & M. College, Fort Collins (surgery); Carroll K. Mingle, ARS, Washington, D.C. (brucellosis); Harry E. Loree, Portales (treatment in atomic war); Donald F. Patterson, U.S. Air Force, Alamogordo, (feline diseases); and John Mason, Santa Fe (public health).

Members of the panels on laboratory aids and virus diseases were Daniel Johnson and Birdsal Carle, Albuquerque; and Lt. Col. Kenneth Burns, San Antonio, Texas. A discussion on small animals was led by Donald F. Patterson; D. L. Cady, El Paso, Texas; and Earl Kraus, Clovis.

Brigadier General Wayne O. Kester, Washington, D.C., president-elect of the AVMA, addressed the group at the luncheon.

s/W. L. BLACK.

South Dakota

State Association.—The annual meeting of the South Dakota Veterinary Medical Association was held on Oct. 4-5, 1956, at the Cataract Hotel, Sioux Falls.

The program included two color films ("Rinderpest" and "With Dog and Gun") and the following speakers and their subjects: Glenn B. Rae, Pierre (disease conditions); W. F. Waddell, Pierre (animal disease eradication activities); D. R. Peterson, Oklahoma A. & M. College, Stillwater (anesthesia, dehorning, agronomic principles); R. P. Azelton, Mobridge (urethrotomy); I. A. Schipper, Fargo, N. Dak. (mucosal disease); F. H. Oberst, Kansas State College, Manhattan (sterility); J. E. Mosier, Kansas State College, Manhattan (small animal practice); M. S. Hofstad, Iowa State College, Ames (respiratory problems); Lewis E. Harris, Norden Laboratories, Lincoln, Neb. (diagnostic methods); and a panel discussion on dispensing problems by Drs. L. E. Harris; R. P. Azelton; D. L. Cotton, Beresford; Harold Severson, Winner; and W. W. Harris, Madison.

s/J. L. NOORDSY, *Secretary.*

FOREIGN NEWS

Brazil

Dr. Jorge Reports on Studies in United States.—Dr. J. Brito Jorge, who returned to Brazil recently from two years of study in the United States, where he spent some time at the veterinary schools at the University of Illinois and Texas A. & M. College, also at the University of Texas Medical Branch in Galveston, sent the AVMA a copy of his official report as a Point-4 grantee and McLaughlin fellow.

Dr. Jorge, a graduate of the veterinary school, Rural University, Rio de Janeiro, also spent some time in the States during World War II. He is now engaged in teaching, research, and extension work at the Animal Biology Institute of the Brazilian University of Agriculture.

DEATHS

★**Warren B. Earl** (GWU '12), 71, Reno, Nev., director of the division of Animal Industry, State Department of Agriculture, since 1931, and secretary of the State Veterinary Examining Board for nearly 20 years, died suddenly on Aug. 18, 1956, following a heart attack.

Born April 9, 1885, at Vernon Center, N. Y., Dr. Earl received his early education in the local schools and his veterinary degree at George Washington University in 1912. He served with the Bureau of Animal Industry for several years and then became field veterinarian with the Nevada State Livestock Board in 1918, a position he held until named director.

He was a member of the Nevada State Veterinary Medical Association and the AVMA, having joined the latter in 1918. He served his state association as secretary from 1931 to 1948 and as president in 1950.

Dr. Earl's wife, the former Laura M. Gilmore to whom he was married in 1918, died several years ago.

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★**Thomas O. Booth** (KCV '16), 66, Springtown, Texas, former chief veterinarian of the Texas Livestock Sanitary Commission and a veteran of both world wars, died on Aug. 9, 1956, at McKinney Hospital of carcinoma of the throat.

Born July 11, 1890, at Independence, Kan., Dr. Booth attended Kemper Military School as a youth and received his veterinary medical training at Kansas City Veterinary College, graduating in 1916. He was commissioned in the Veterinary Corps in 1917, served as division veterinarian of the Thirty-Sixth Division at Camp Bowie, Texas, and overseas with the A.E.F., being discharged June 30, 1919, with the rank of major. He was a member of the Organized Reserves for several years and was recalled to active duty early in World War II.

Between wars, Dr. Booth was assistant state veterinarian in Oklahoma for a time and then joined the Texas Livestock Sanitary Commission; he resigned as chief veterinarian in 1947.

Dr. Booth was a member of the Texas State Veterinary Medical Association (president in 1930) and the AVMA which he joined in 1922.

Surviving are his widow, the former Leta P. Lessinger; a daughter, two sons, a brother, and 11 grandchildren.

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★**Vincent G. Bromwell** (ISC '18), 60, Center Point, Iowa, died July 15, 1956. Dr. Bromwell, a general practitioner, had served in the Veterinary Corps, U.S. Army, in World War I and was active in civic affairs in his community. He was a member of the Iowa State and Eastern Iowa Veterinary Medical Associations and of the AVMA which he joined in 1919.

Otis G. Button (WSC '16), Tacoma, Wash., died in June, 1956. Dr. Button had retired from practice some years ago.

★**Cornelius Cann** (CVC '91), 95, died at Frankfort, Ky., on Aug. 16, 1956, at the age of 95. Burial was at Lexington. For about 60 years, prior to his retirement four years ago, he specialized in equine practice. He was especially adept at firing horses and his services were in demand in all parts of the country. Dr. Cann's survivors are a daughter, Mrs. Stanley Hudson, of Frankfort, with whom he made his home in recent years, and two grandchildren.

★**Robert L. Cornell** (KSC '47), 31, Whittier, Calif., died suddenly on July 25, 1956, after a short illness. Dr. Cornell engaged in small animal practice for a short period following his graduation. For the past several years, he was a member of the Los Angeles County Livestock Department, and was held in high regard by his many friends and associates. Dr. Cornell was a member of the Southern California Veterinary Medical Association and of the AVMA. He is survived by his widow and a son.

Lawrence C. Daugherty (IND. '04), Sheridan, Ark., died June 22, 1956. Dr. Daugherty had practiced in Kissimmee, Fla., for many years, prior to his retirement. His widow and a son survive.

★**James C. Exline** (MCK '05), 80, Santa Barbara, Calif., died July 14, 1956. Soon after graduation, Dr. Exline entered the service of the BAI, U.S.D.A., and was advanced to the position of inspector in charge, which he held until his retirement in December, 1946. He was successful in directing the eradication of bovine tuberculosis and scabies. Througout his long service in the BAI, he was recognized as a successful leader in all the projects to which he was assigned. Dr Exline served in the Spanish-American War, in World War I, and with the chemical and intelligence divisions of the U. S. Army. He was a member of the Military Order of the World Wars, American Legion, the AVMA, and was a 32nd degree Mason. His survivors include his widow, two daughters, and a grandson.

John R. Friedline (GR '16), 61, Sarasota, Fla., died in May, 1956. Dr. Friedline had practiced in Woodlake, Minn., until his retirement in 1949. His widow survives.

★**John F. Hemphill** (KCV '07), 89, Clay Center, Kan., died July 11, 1956. Dr. Hemphill had practiced in Clay Center since 1907. He was a member of the AVMA for 44 years. His survivors include his widow, four sons, and two grandchildren.

John H. McGilvray (ISC '42), Denver, Colo., died June 30, 1956. Dr. McGilvray was a major in the Veterinary Corps, U. S. Army.

★**Alexander T. McIntyre** (OVC '12), 74, Brown City, Mich., died instantly, on July 11, 1956, when the car he was driving collided with a train. Dr. McIntyre was a member of the Michigan State Veterinary Medical Association and of the AVMA which he joined in 1923. His widow survives.

★**Ray S. Robinson** (MCK '14), 65, Pierre, S. Dak., died July 14, 1956. Dr. Robinson was a member of the South Dakota Veterinary Medical Association and of the AVMA.

Archie L. Schenck (GR "18), 72, Butler, Pa., died July 13, 1956. Dr. Schenck had been in poor health for some time. He is survived by a son, three daughters, and five grandsons.

Lloyd J. Smith (KCV '15), 66, Fairbury, Neb., died July 3, 1956. Dr. Smith was a general practitioner.

★**Patrick J. Wilkinson** (CVC '94), 93, Oconomowoc, Wis., died March 8, 1956. Dr. Wilkinson had been a member of the AVMA for 30 years.

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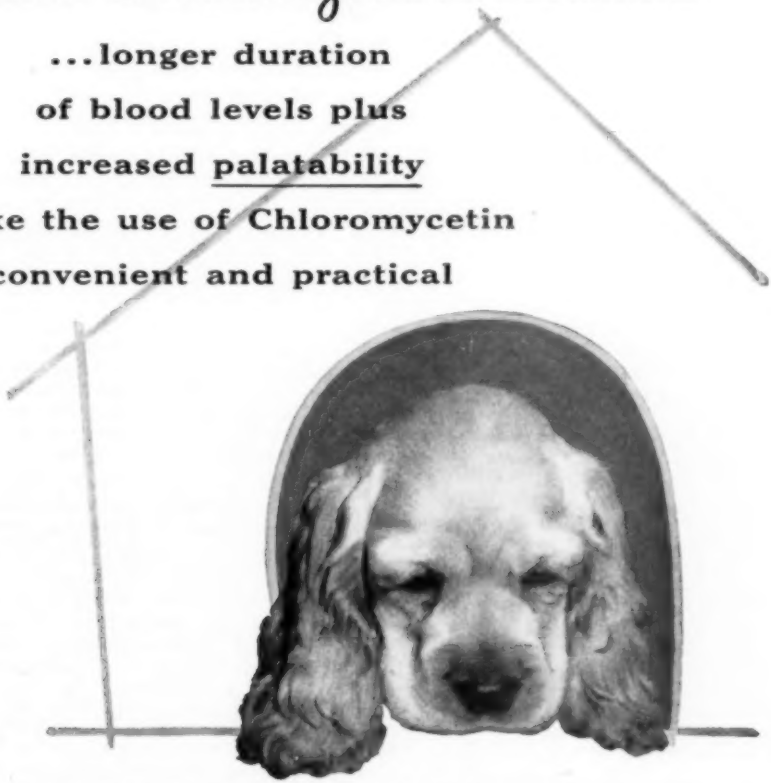

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ORGANIZATION SECTION

Veterinarians Added to OASI Programs

The Social Security bill, bringing self-employed veterinarians into the Old Age and Survivors' Insurance program for the first time, was signed by President Eisenhower on August 1, after clearing Congress in the final hours of the 84th session. It added about 200,000 self-employed persons to the program, including veterinarians, dentists, lawyers, and osteopaths. The only exempt professional group remaining are the physicians.

SOCIAL SECURITY TAXES

Veterinarians will pay taxes of 3 per cent on the first $4,200 of annual net earnings, or $126.00, the first assessment to be filed with the 1956 income tax return in early 1957. The starting date for compulsory credit toward retirement payments and other benefits under the program would be Jan. 1, 1956.

The scheduled rate for future taxable years are as follows:

1957 through 1959	3⅜%
1960 through 1964	4⅛%
1965 through 1969	4⅞%
1970 through 1974	5⅝%
After 1974	6⅜%

OASI coverage for the veterinary profession is compulsory. Whether or not the veterinarian wants to participate in the program available under OASI makes no difference. He must pay the full amount of specified self-employment taxes on his annual earnings.

Each veterinarian should have a social security account number. If he does not have one, he should apply for a social security card at his local social security office. If there is no social security office nearby, ask for an application blank (form SS-5) at the post office, fill it out, and mail to the nearest Social Security office. If he did have one, but can not find it, he should apply for a duplicate card. The individual's social security account number must be listed on his annual tax returns.

BECOMING ELIGIBLE FOR BENEFITS

The formula for determining eligibility for benefits is quite complicated. It is based on the number of calendar quarters in which the individual was liable for, and paid, a social security tax.

A minimum of six quarters of coverage is required in every case before the tax payer or his dependents are eligible for benefits.

A special provision of the 1956 amendments provides that self-employed veterinarians who have reached their 65th birthday or who will be 65 years of age before July 1, 1957, may qualify for benefits by that date if they have six quarters of coverage. There is no age limit for starting quarters of coverage.

Following are the requirements necessary to qualify a self-employed veterinarian for monthly retirement benefits:

1) If his 65th birthday has already occurred, or will occur before July 1, 1957, a self-employed veterinarian will qualify for OASI benefits by earning at the rate of at least $400 per year between Jan. 1, 1956, and July 1, 1957.

2) If his 65th birthday will occur after July 1, 1957, the self-employed veterinarian will qualify for OASI benefits by earning at the rate of at least $100 per quarter ($400 per year) during one of the following periods of time:

a) between Jan. 1, 1956, and his 65th birthday;

b) for one half the time between Jan. 1, 1951, and his 65th birthday;

c) for ten years between Jan. 1, 1956, and his 65th birthday; or

d) for ten years between his 21st birthday and his 65th birthday.

Questions about the situation of any particular individual can best be answered by representatives of the Social Security Administration. Future issues of the JOURNAL will carry a series of questions and answers in this column pertaining to social security based on information furnished by the local Social Security Administration.

WOMEN'S AUXILIARY

Acting President—Mrs. Alfred E. Coombs, P.O. Box 174, Skowhegan, Maine

Secretary—Mrs. F. R. Booth, 3920 E. Jackson Blvd., Elkhart, Ind.

Canada Auxiliary.—At the annual meeting of the Women's Auxiliary to the Canadian Veterinary Medical Association, held on July 20, 1956, the following officers were elected for the ensuing term: Mrs. G. J. MacDonald, Assiniboine, Sask., president; Mrs. W. J. Rumney, Aldershot, Ont., first vice-president; Mrs. George Fisher, Charlottetown, P. E. I., second vice-president; Mrs. J. J. Andrich, Saskatoon, Sask., third vice-president; Mrs. E. N. Anderson, Boniface, Man., secretary; and Mrs. J. W. Goin, Wetaskiwin, Atla., treasurer.

s/Mrs. E. N. Anderson, *Secretary.*

• • •

South Dakota Auxiliary.—The Women's Auxiliary to the South Dakota Veterinary Medical Association held a meeting on Oct. 4-5, 1956, at the Cataract Hotel in Sioux City, in conjunction with the annual meeting of the South Dakota Veterinary Medical Association.

The women's program included a luncheon, banquet, and a bridge and social hour.

s/Mrs. George Chaffee, *Secretary.*

NO. 3 OF A SERIES:

Controlling the worms in cattle with new Pheno-Fortified Salt

Q. Do some modern management practices tend to intensify the worm problem?

A. Yes, pasture irrigation, for one. In addition to providing more feed, it also provides optimum conditions for parasite growth—even temperature and moisture at the base of plants, where parasites thrive. And the fact that irrigated pastures carry more animals, and are often used for susceptible young stock, makes them an even more important consideration in the new approach to worm control.

Q. Why has 2-way worm control with phenothiazine proved most practical?

A. Because it controls both the immediate worm problem and that of reinfection. Here's how it works: After veterinary diagnosis, phenothiazine, as a worming dose, is used to knock out adult worms. Then "pheno," in a suitable carrier, is fed daily in small amounts . . . to suppress production of viable eggs that contaminate pastures. This assures the continuing protection needed by young dairy animals, beef stock, feeder steers and sheep. Programs like this are now recommended by 17 states, as well as by other groups who have recognized the growing seriousness of today's worm problem. Note: phenothiazine is the parasiticide that kills a wider range of parasites than any other drug of its kind in the veterinary field.

Q. Why is phenothiazine in salt especially effective for low-level feeding?

A. Because salt, an ideal carrier for "pheno," is needed every day of the year by livestock; and because salt fits any feeding program. One of the best commercial products for this type program is Sterling Green'salt —produced by the International Salt Company. Fed free choice or as the salt in feed (at the rate of 1%), Green'salt has proved ideal for low-level control. It contains salt, 10% "pheno," and the six essential trace minerals: iodine, cobalt, iron, copper, manganese and zinc—as well. Daily intake of Green'salt per head should be about two tablespoons for adult cattle, one-half tablespoon for sheep. This feeding should be continuous, following the worming dose.

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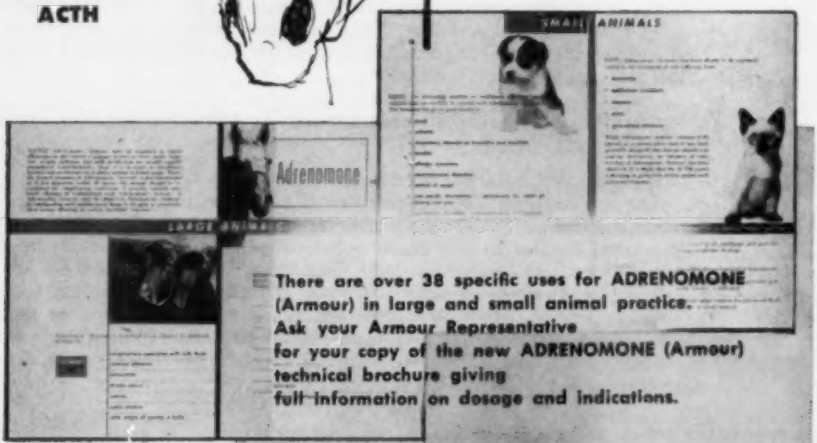

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COMING MEETINGS

American Veterinary Medical Association. Annual meeting. Municipal Auditorium, San Antonio, Texas, Oct. 15-18, 1956. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Veterinary Symposium on Dogs. Kankakee Civic Auditorium, Kankakee, Ill., Oct. 24, 1956. Mr. Harry Miller, Gaines Dog Research Center, 250 Park Ave., New York 17, N. Y., director.

Interstate Veterinary Medical Association. Annual meeting. Martin Hotel, Sioux City, Iowa, Oct. 30-31, 1956. K. W. Smith, 510 W. 19th St., Sioux City, Iowa, secretary.

Mississippi Valley Veterinary Medical Association. Annual meeting. Hotel Pere Marquette, Peoria, Ill., Nov. 7-8, 1956. William L. Beer, 612 N. College Ave., Aledo, Ill., secretary.

Midwest Small Animal Association, annual meeting, and American Animal Hospital Association, regional meeting. Hotel Burlington, Burlington, Iowa, Nov. 18-19, 1956. J. Porter Coble, 2828 S. MacArthur Blvd., Springfield Ill., secretary, Midwest Small Animal Association.

U. S. Livestock Sanitary Association. Annual meeting. Morrison Hotel, Chicago, Ill., Nov. 28-30, 1956. R. A. Hendershott, 33 Oak Lane, Trenton 8, N. J., secretary.

Animal Care Panel. Annual meeting. Morrison Hotel, Chicago, Ill., Nov. 29-30, 1956. Robert J. Flynn, P.O. Box 299, Lemont, Ill., secretary.

Nebraska Veterinary Medical Association. Annual meeting. Hotel Lincoln, Lincoln, Dec. 3-5, 1956. W. T. Spencer, 1250 North 37th St., Lincoln, secretary.

New York State Veterinary College. Annual conference for veterinarians. New York State Veterinary College, Cornell University, Ithaca, Jan. 2-4, 1957, W. A. Hagan, dean.

Oklahoma Veterinary Medical Association. Annual meeting. Huckins Hotel, Oklahoma City, Jan. 14-15, 1957. M. N. Riemenschneider, 122 State Capitol Building, Oklahoma City, secretary.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Iowa, Jan. 22-24, 1957. F. B. Young, Waukee, Iowa, secretary.

California State Veterinary Medical Association. Midwinter conference. School of Veterinary Medicine, University of California, Davis, Jan. 28-30, 1957. Charles S. Travers, 3004 16th St., San Francisco, executive secretary.

Louisiana State University. Annual conference for veterinarians. Pleasant Hall, Louisiana State University, Baton Rouge, Jan. 29-30, 1956. W. T. Oglesby, Department of Veterinary Science.

Minnesota State Veterinary Medical Association. Annual meeting. Radisson Hotel, Minneapolis, Feb. 4-6, 1957. B. S. Pomeroy, 1443 Raymond Ave., St. Paul 8, secretary.

Foreign Meetings

Asociación Médico Veterinaria de Puerto Rico. Inter American veterinary medical symposium. Caribe Hilton Hotel, San Juan, Nov. 30-Dec. 2, 1956. O. A. López-Pancheco, P.O. Box 155, Hato Rey, Puerto Rico, chairman.

(Continued on p. 30)

JOURNAL OF PARASITOLOGY

Official organ of American Society of Parasitologists

A medium for the publication of papers on Animal Parasites (Protozoa, Helminthes, Arthropoda).

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GEORGE R. LA RUE

CHAIRMAN, EDITORIAL COMMITTEE

JOURNAL OF PARASITOLOGY

U.S. Bureau of Animal Industry,

Zoological Division
Beltsville, Maryland

Dr. Huffman Joins Armour Laboratories

Dr. Earl F. Huffman has joined the Armour Veterinary Laboratories, Kankakee, Ill., as manager of professional relations.

Dr. Huffman received his D.V.M. degree from Michigan State University. He entered general practice and joined the U.S. Department of Agriculture, Animal Disease Eradication Branch, where he did diagnostic and supervisory work. He also did graduate work at the University of Wisconsin.

Dr. Earl F. Huffman

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(COMING MEETINGS—*continued from p. 29*)

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the first Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Pima County Veterinary Medical Association, the third Wednesday of each month in Tucson. E. T. Anderson, 8420 Tanque Verde Rd., Tucson, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Bay Counties Veterinary Medical Association, the second Tuesday of each month. E. Paul, Redwood City, Calif., secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. Wilfred Pimentel, 3455 S. Elm Ave., Fresno, Calif., secretary.

Association of East Bay Veterinarians, bimonthly, the fourth Wednesday. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. T. D. Harris, San Mateo, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

(*Continued on p. 31*)

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5000 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the second Friday of each month, time and place specified monthly. James B. Murphy, Eustis, Fla., secretary.

Jacksonville Veterinary Medical Association, the second Thursday of each month, time and place specified monthly. George F. Yopp, 4644 Main St., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. Harold A. Tennant, Atmore, Ala., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. Ross E. Evans, 5215 S. Dixie Highway, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. D. Stoddard, 6432 S. W. 8th St., Miami, Fla., secretary.

Suwannee Valley Veterinary Association, the third Friday of each month, at the Thomas Hotel, Gainesville, Fla. R. C. Mann, Rt. 1, Box 37, Ocala, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month, except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association the third Thursday of each month. W. E. Sharp, Union City, Ind., secretary.

IOWA—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. H. V. Henderson, Reinbeck, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Winneshiek Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

(*Continued on p. 34*)

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KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of the month (except July and August) at the Sheraton Hotel, Spring Ave. and Lindell Blvd. Allen B. Shopmaker, 136 N. Meramec, Clayton 5, Mo., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at alternating hospitals. W. F. Noland, 7504 Metcalf, Overland Park, Kan., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month at Exchange Hall, ninth floor, Livestock Exchange Bldg., 1600 Genessee St., Kansas City, Mo. Busch Meredith, 800 Woodswether Rd., Kansas City 5, Mo., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel in Greensboro. J. W. Peace, High Point, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month. Wm. Allen Potts, 401 W. James St., Mount Olive, secretary.

Piedmont Veterinary Medical Association, the last Friday of each month at 7:00 p.m. in Mull's Motel in Hickory,

(*Continued on p. 35*)

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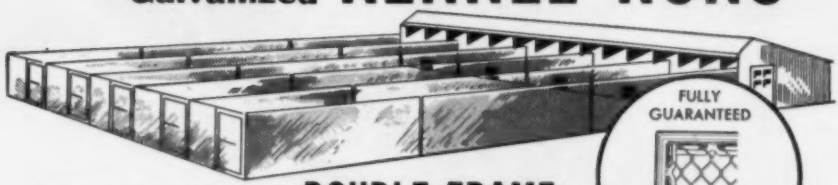

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(COMING MEETINGS—*continued from p. 34*)

N. Car. W. W. Dickson, Box 1071, Gastonia, N. Car., secretary.

Redmont Veterinary Medical Association, the last Tuesday of the month. J. L. Innes, 9 Tampa Ave., West Asheville, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month. James M. Brown, 2818 W. Britton Rd., Oklahoma City, secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, Va., secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Tuesday of each month in the Trinity Episcopal Church, 8th and James St., Seattle, Wash. P. R. Des Roniers, 5308 2nd Ave., N. W., Seattle 7, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. L. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W., Huntington, W. Va., secretary.

WISCONSIN—Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. George F. Lynch, 201 West Devon St., Milwaukee 17, Wis., secretary.

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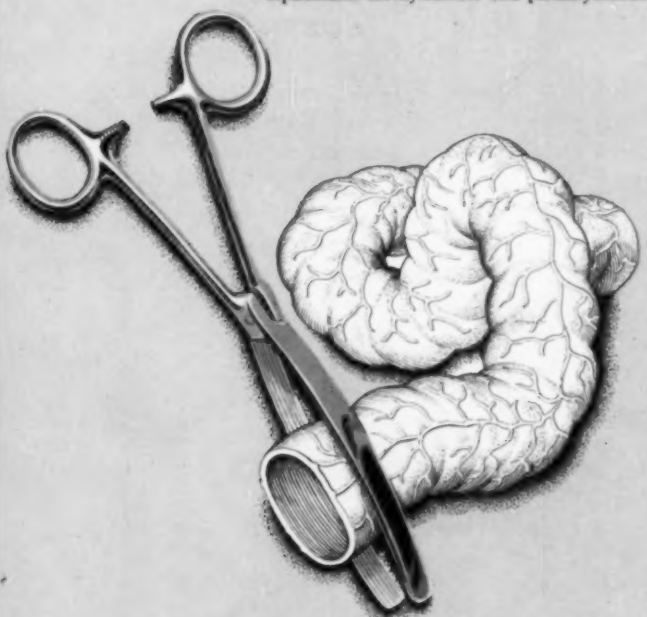

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REFERENCES

1. Koger, R. B.: Vet. Med. *50*:713, 1955.
2. Chambers, E. E.: North Am. Veterinarian *37*:105, 1956.
3. Shaw, J. C.; Gessert, R. A., and Chung, A. C.: North Am. Veterinarian *36*:918, 1955.
4. Morris, R. C., and Hall, C. E.: J.A.V.M.A. *128*:132, 1956.

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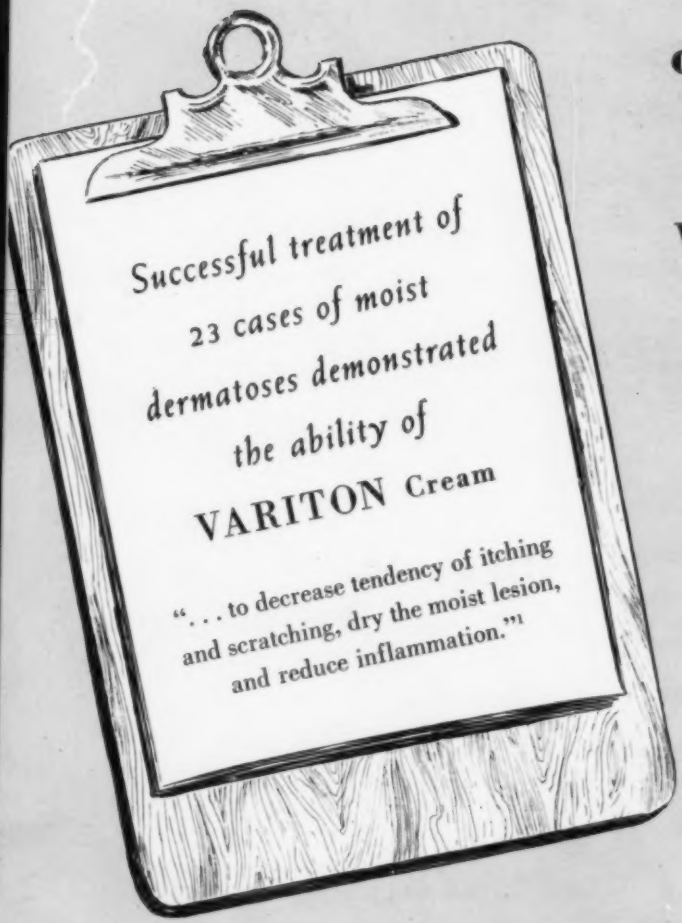

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1. Phillips, L. R.: Vet. Med. *51*:232, 1956.

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Nationally established ethical manufacturer distributor of veterinary pharmaceuticals offers immediate opportunity for man qualified and/or experienced in the production of veterinary pharmaceuticals. Address "Box W 5," c/o JOURNAL of the AVMA.

Graduate of Ontario Veterinary College wanted for small animal practice in Toronto. Write giving full details and remuneration desired to Dr. J. N. Stratas, 132 Danforth Ave., Toronto 6, Ontario.

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(Continued on p. 38)

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(CLASSIFIED ADS—*continued from p. 37*)

Veterinarian wanted—$5,702-$6,242 (The Civil Service Commission has approved a new rate of $6,505 to $7,225 per year, subject to the concurrent approval of the Board of Supervisors). New position in the Wayne County Health Department located 17 miles from downtown Detroit. Meat inspection, dairy farm controls, coordination of rabies control program, and enforcement of County and State quarantine measures. Not over 51 years of age and eligible for a Michigan Veterinary License. Contact Morton Hilbert, Chief of Engineering and Sanitation, Wayne County Health Department, Eloise, Mich.

Wanted—Positions

Position wanted in small animal or mixed practice; graduate, 1954; licensed in Washington, Oregon, California. Will separate from service Jan. 1, 1957. Address Capt. Alonzo E. Howard, 5010th U.S.A.F. Hospital, A.P.O. 937, Seattle, Wash.

Experienced general practitioner, age 33, married, desires position with ethical pharmaceutical company as drug salesman or in the field of professional relations. Address "Box W 27," c/o JOURNAL of the AVMA.

Remittance must accompany advertisement

Veterinarian desires assistantship busy small animal hospital; some experience. Licensed Ohio, Maryland, Washington, D.C. Available immediately. Address "Box W 28," c/o JOURNAL of the AVMA.

Wanted—Practices

Want to lease profitable small animal practice, preferably with option to buy, in northern Middlewest; 27, married, experienced, AVMA-approved school. Will consider any good location. Address "Box W 4," c/o JOURNAL of the AVMA.

(*Continued on p. 39*)

This exhibit in lard has appeared at state fairs in the Middlewest and is scheduled for the American Royal Livestock Show and the International Livestock Exposition in October and November, respectively.

(CLASSIFIED ADS—*continued from p. 38*)

Hard working veterinarian with 10 years of experience in mixed practice desires partnership or purchase of established practice in eastern Iowa or northern Illinois. Address "Box W 22," c/o JOURNAL of the AVMA.

Graduate, 1955, wants to purchase predominantly small animal practice within 1½ hours driving time of New York City. Adequate capital available. Address "Box W 24," c/o JOURNAL of the AVMA.

Small animal practice wanted by capable, hard working veterinarian to purchase or lease; also consider partnership arrangement. Age 30, married; consider any good location. Address "Box W 26," c/o JOURNAL of the AVMA.

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Mixed practice, 5 years old, for sale, east central Illinois; modern masonry home and hospital combination, tile office, surgery, etc. Building, 3½ years old, on 1½ acres of land, main highway. No close competition; 65% large, 35% small animals with growth in both. A one-man practice grossing over $20,000. Address "Box W 13," c/o JOURNAL of the AVMA.

Practice for sale on Guam; only civilian veterinarian. Large house, office, kennels, plus 30 furnished apartments. Monthly income, $3,000; complete price $35,000. Address Dr. C. W. Hansen, P.O. Box 855, Agana, Guam.

Established small animal hospital for sale; modern basement hospital, drugs, and equipment and 6-room brick home. Address Dr. Fred P. Mursko, 1010 Sheridan Blvd., Denver 14, Colo.

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Large animal practice for sale in Middlewest; new ranch home, office, drugs, equipment. Average yearly gross, $46,000; $25,000 will handle. Address "Box W 23," c/o JOURNAL of the AVMA.

Excellent dairy practice for lease or sale in Missouri; good climate, city, and recreation. Personality and capability of work volume necessary. Give full qualifications and ambitions first letter. Address "Box W 25," c/o JOURNAL of the AVMA.

For lease in Phoenix, 150 new boarding kennels, excellent income, great demand for veterinary service. Responsible man can double gross. Address Bramley Animal Hospital, Scottsdale, Ariz.

Mixed practice for sale; long-established, fully equipped small animal hospital and 9-room house included. Address Dr. E. Garner, E. Main St., Mendham, N. J.

Miscellaneous

Pregnancy diagnosis in mares—45th to 150th day. Request mailing tubes; $7.00, 2 or more, $6.00 each. Pregnancy Diagnostic Laboratories, Dysart, Iowa.

(*Continued on p. 42*)

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STERANE®
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FOR THE CONTROL OF SPASTIC DISORDERS in small animals

TRASENTINE relieves both neurogenic and myogenic spasm of the smooth musculature. It possesses properties of atropine and papaverine without the side effects usually following atropine. It has low toxicity and is non-cumulative.

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Today's First and Only Pellet Implant Developed Exclusively for Steers!

• Scalpel-type knife makes quick, clean incision. Implanter inserts the Synovex implant between skin and cartilage of ear. Each plastic cartridge contains a total of 1 gm. Progesterone and 20 mg. Estradiol Benzoate in 6 pellets which constitutes one Synovex implant.

• Synovex is available in 10-implant and 100-implant packages. Package containing implanter and knife is sold separately.

...gives your clients

higher weight gains
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SYNOVEX is a registered Squibb trademark.

1 Insert cartridge in implanter.

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3 Make incision with special knife.

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SYNOVEX IS EASY TO USE. Special plastic cartridge containing one implant (six pellets) of Synovex fits easily into shank of implanter—implanter inserts Synovex between skin and cartilage of ear in single operation. Scalpel-type knife makes quick, clean incision. Synovex is available in 10-implant and 100-implant packages and is recommended for animals weighing from 400 to 1000 lbs., for use during the last 60-150 days of finishing. Synovex goes to work in minutes after implant, and results in marked increase in appetite within seven days. Implant remains effective for 150 days, *boosting weight gains an average of ½ lb. per day extra over controls.*

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(1) R. J. Deans, W. J. Van Arsdell, E. P. Reineke and L. J. Bratzler (Michigan Agricultural Experiment Station): The Effect of Progesterone-Estradiol Implants and Stilbestrol Feeding on Feed Lot Performance and Carcass Characteristics of Steers, *Jl. of Animal Science* (In Press).

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(CLASSIFIED ADS—*continued from p. 39*)

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Exclusive Publication.—Articles submitted for publication are accepted with the understanding that they are not submitted to other journals, which is ethical publication procedure.

Manuscripts.—Manuscripts, including **footnotes, references,** and **tables,** must be typewritten, **double-spaced,** on 8½- by 11-in. bond paper, and the **original,** not the carbon copy, submitted. One-inch margins should be allowed on the sides, with 2 in. at top and bottom. Articles should be concise and to the point. Short, simple sentences are clearer and more forceful than long, complex ones.

Illustrations.—Photographs should be furnished in glossy prints, and of a size that will fit into the JOURNAL of the American Veterinary Medical Association with a minimum of reduction. Photomicrographs which can not be reduced should be marked for cropping to 1-column or 2-column width. Identifying marks within the photomicrographs, such as arrows, letters, or numbers, should be clearly marked with black India ink or white opaque ink to insure good reproduction and must be large enough to stand reduction, if necessary. Drawings, graphs, and charts should be made clearly and accurately in India ink on white paper and a glossy print of them submitted when possible. Numbers or letters appearing on graphs or charts should be large enough to allow for any reduction necessary for the chart or graph to fit JOURNAL pages. Blue lines in graph paper drop out in reproduction; therefore, if lines are required they must be drawn in black ink. All illustrations should bear the name of the author and the illustration number on the back.

Tables.—Tables should be simple and typed **double space.** Complex tables are not conducive to perusal. It is wiser to summarize complex material than to tabulate it.

References.—References should be typed **double space** and should be prepared in the following style: name of author, title of article, name of periodical with volume, year, and page numbers. References to journals not commonly known should give the complete name of the periodical, and where published so that they may be added to our reference files. When books are cited, the name of publisher, location, edition, and year should be given.

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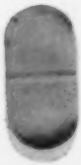

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Shaker-Top Bottle

HAVOSEPT'S activity is not affected by the temperature or hardness of water. The pH of Havosept is adjusted to that of the skin. It is free of harmful alkalis and fatty acids. Thus, the veterinarian with intolerance to alkali soaps is not adversely affected by using Havosept.

HAVOSEPT is excellent for pre-operative scrubbing of both surgeon and patient. It prevents regeneration of bacterial flora. These antiseptic and protectant properties are important to you in prevention of wound contamination, and transmission of bacteria to you, the operator. Havosept is ideal as a lubricant for vaginal and rectal examination as well as for obstetrical procedures. This multi-purpose detergent also overcomes objectionable odors.

HAVER-GLOVER
Laboratories

SEE HAVER-GLOVER MESSENGER FOR FORMULA AND PRICE

WHICH LOCKHART RABIES VACCINE SHOULD YOU USE?

CHICK EMBRYO ORIGIN

Modified Live Virus Rabies Vaccine prepared from the Flury strain of rabies virus, modified in virulence by passage in chick embryos.

This modified strain has demonstrated high immunizing ability in extensive field trials.

For use only in the immunization of healthy dogs against rabies.

OVINE ORIGIN

A sterile, neutral, phenolized 20% suspension prepared from the brain and spinal cord of animals showing typical symptoms of rabies following subdural injection of the fixed virus of rabies.

For the immunization of dogs and other animals against rabies.

YOU MAKE THE CHOICE!

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Lockhart now offers both vaccines for your consideration . . . you make the choice!

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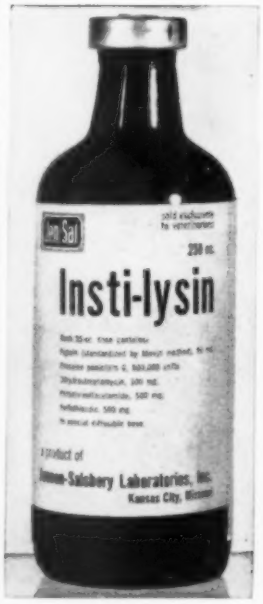
Jen-Sal



each 25 cc. dose
contains
50 mg. Papain
(pure crystalline)
500,000 units
Procaine penicillin G,
100 mg.
Dihydrostreptomycin,
500 mg.
Phthalylsulfacetamide,
500 mg.
Sulfathiazole,
in special
diffusible
base

pure crystalline Papain
makes Jen-Sal

INSTI-LYSIN

the superior therapy in acute mastitis

The superior effectiveness of Insti-lysin as therapy in acute mastitis and other conditions requiring effective debridement has been well established by continuous field usage.

Pure crystalline papain gives Insti-lysin twice the proteolytic activity of crude papain, without irritation to normal tissue, and without systemic or generalized reactions.

Antibiotics and sulfonamides assure broad bacterial coverage and maximum antibacterial activity. As an udder infusion, Insti-lysin rises rapidly to the site of infected tissue in its special, diffusible base.

Insti-lysin is available in 3 different packages to meet the specific needs of individual practices; 250 cc. vial for professional use; display carton of 12—28 cc. mastitis syringes in individual dispensing cartons; package of 12—5 cc. syringes in dispensing cartons for small animal clients.

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